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APRIL 1956

THURSTON SCOTT WELTON, M.D., EDITOR

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The American Association
for the
Surgery of Trauma

SIXTY-FIFTH YEAR OF PUBLICATION

APRIL 1956

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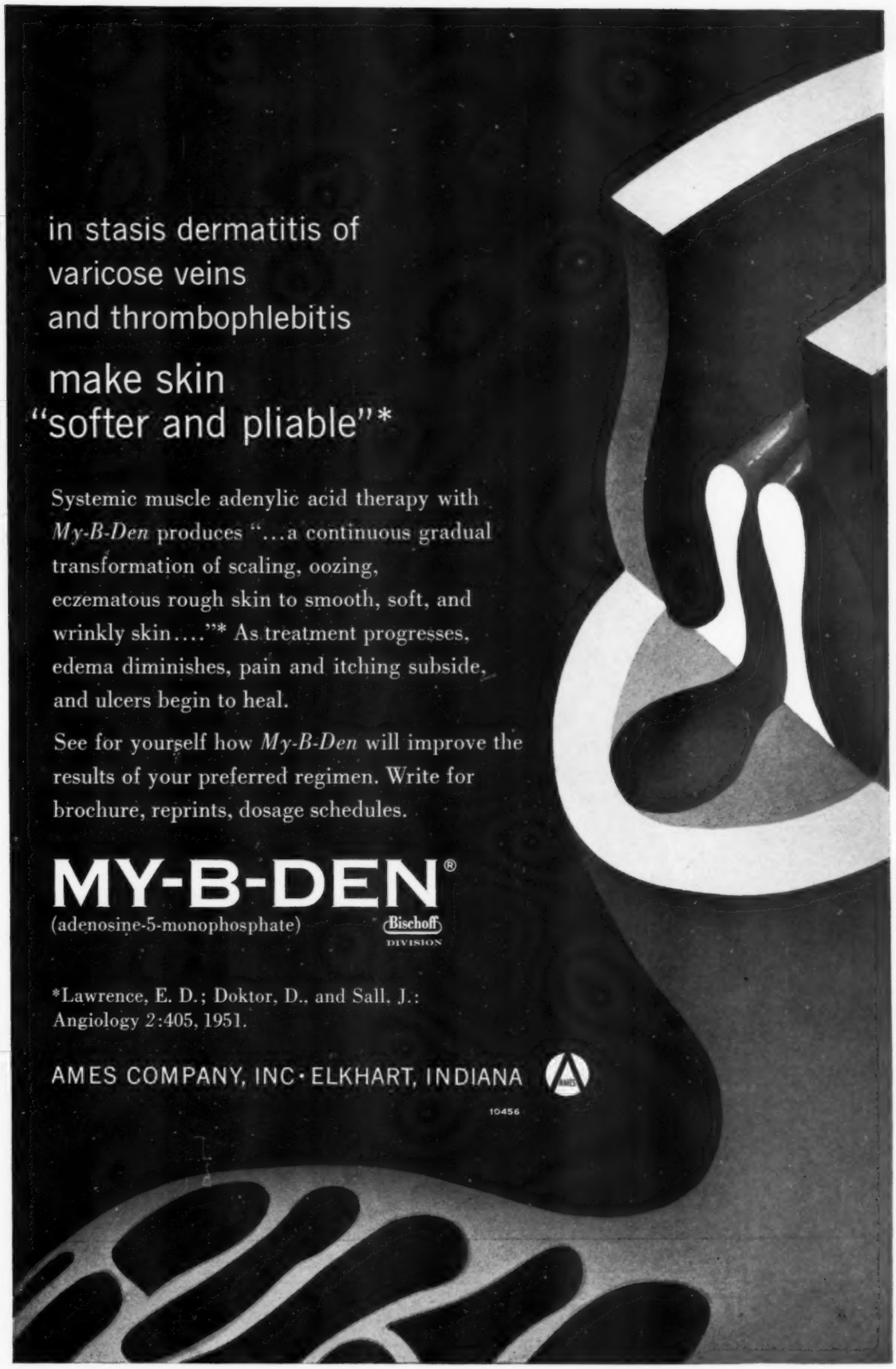

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1. Eckenhoff, J. E.; and Dripps, R.D.: Anesthesiology, 15:681, Nov., 1954. 2. Sokoloff, Louis; King, B. D.; and Wechsler, R. L.: Med. Clin. North America, 38:499, Mar., 1954.

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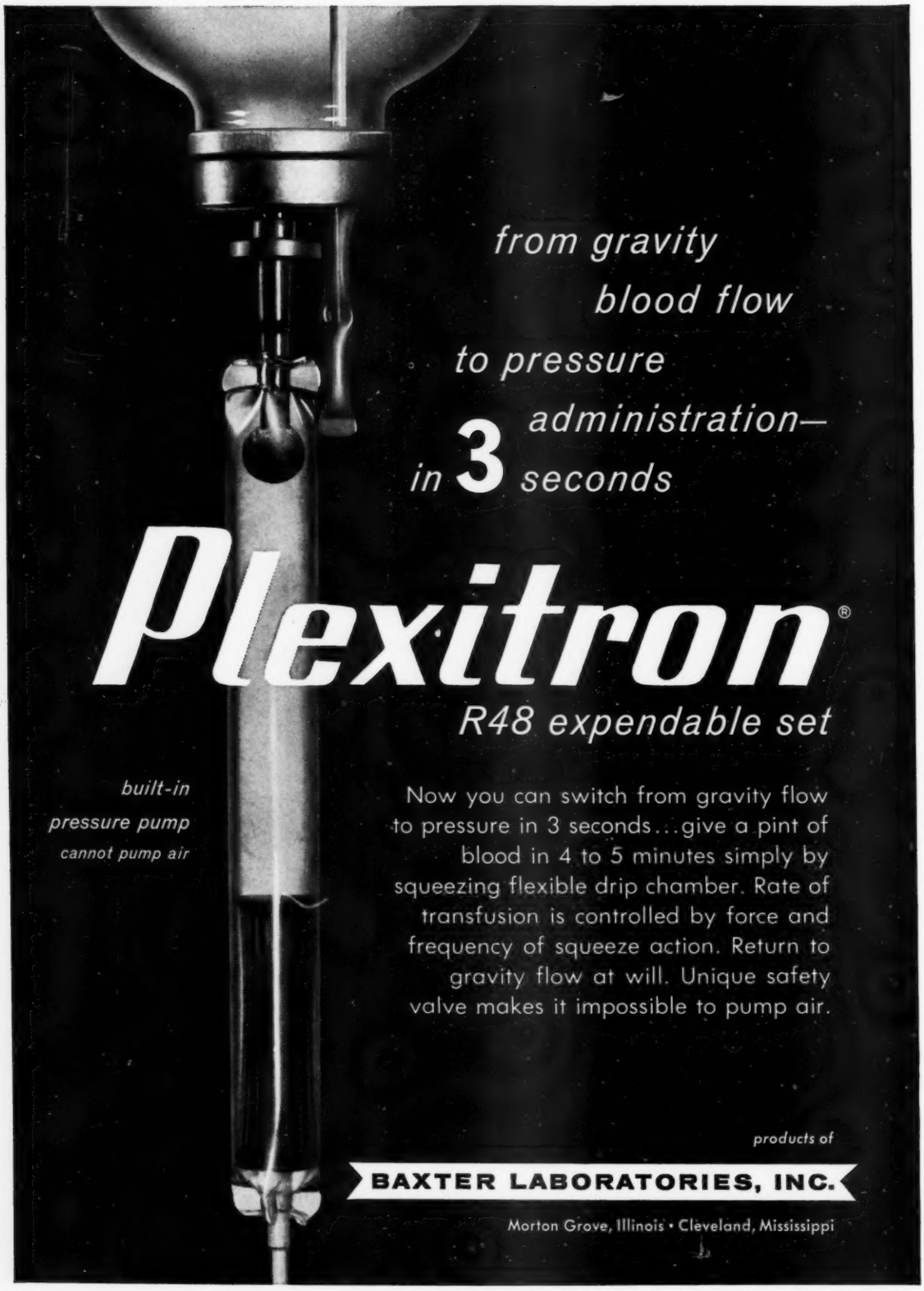
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A Laboratory and Clinical Report on Adrenosem® Salicylate

(BRAND OF CARBAZOCHROME SALICYLATE)

History

The first investigation of a hemostat with an action comparable to Adrenosem Salicylate was made by Derouaux and Roskam[1] in 1937. They reported that an oxidation product of adrenalin, adrenochrome (which has no sympathomimetic properties), has prompt hemostatic activity.

It was further found that various combinations of adrenochrome, notably the oxime and semicarbazone, produced stable solutions. But, these were so slightly soluble that sufficient concentration could not be obtained for practical therapeutic use. By combining these adrenochrome compounds in a sodium salicylate complex a stable, soluble form can be obtained. This complex has been given the generic name, carbazochrome salicylate, and is supplied under the trade name Adrenosem Salicylate.

Roskam, in his study entitled "The Arrest of Bleeding,"[2] enumerates "the drugs whose efficaciousness as hemostatics have been proved by accurate methods in experimental animals and in healthy men as well. . . . One is the monosemicarbazone of adrenochrome [Adrenosem Salicylate]."

Chemistry

Adrenosem Salicylate is a synthetic chemical. The full chemical name is adrenochrome monosemicarbazone sodium salicylate complex.

Pharmacology

Although it is chemically related to epinephrine, Adrenosem Salicylate has no sympathomimetic effects. It does not alter blood components, nor does it affect blood pressure or cardiac rate.[2-7]

(* U.S. Patent 2,581,850)

Sherber, in an early study,[3] concludes that Adrenosem Salicylate* "is a potent antihemorrhagic factor in those conditions in which the integrity of the smaller vessels is interrupted, and is superior to any similar material that is now available."

He continues, "From our experience it appears that adrenochromazone complex is indicated in preventing vascular accidents incident to hypertension; in maintaining small vessel integrity; in the preoperative preparation where oozing from a vascular bed is anticipated, as in tonsillectomies, adenoidectomies and prostatectomies; and as an adjunct in the treatment of bleeding from such surgical procedures."

Adrenosem Salicylate may be administered simultaneously (but separately) with any type of anesthetic, anticoagulant, or vitamin K and heparin.

A Unique Systemic Hemostat

Clinical investigators[2-7] are in agreement that Adrenosem Salicylate controls bleeding and oozing by decreasing capillary permeability and by promoting the retraction of severed capillary ends. It aids in maintaining normal capillary integrity by direct action on the intercellular "cement" in capillary walls. The interesting work of Fulton[8] confirms this. Adrenosem Salicylate, since it is not a vasoconstrictor, has no effect on large severed blood vessels and arterioles.

Adrenosem Salicylate is being used both prophylactically and therapeutically in thousands of hospitals, and in virtually every type of surgical procedure. It has also proved most useful in dental surgery.[7]

Owings reported on the use of Adrenosem Salicylate in controlling postoperative adenoid bleeding in 102 cases.[4] "We have used 2½ mg.

(½ ampule) intramuscularly, 15 minutes before anesthesia for children and 5 mg. (1 ampule) for adults." In only one patient did bleeding occur. Three others showed red blood from the nose and mouth. These patients "were then given 5 mg. intramuscularly, with prompt and complete control. We have also noticed that bleeding stopped more promptly on the operating table."

This is a 1% incidence of postoperative bleeding using Adrenosem Salicylate preoperatively, compared to an incidence of 10% postoperative bleeding in all cases taken from previous records, without Adrenosem Salicylate medication.

Peele reports on the use of Adrenosem Salicylate in treating 178 patients with 24 different conditions.[5] The drug was first used to control postoperative hemorrhage from the adenoid region. He adds: "The results were so dramatic that since that date [1953] Adrenosem Salicylate has been used postoperatively to reduce bleeding from all otolaryngologic and bronchoesophagolic procedures, to treat postoperative hemorrhage from the tonsil and adenoid regions, and to treat selected cases of epistaxis."

The effectiveness of Adrenosem Salicylate in controlling bleeding and oozing in 330 patients is reviewed by Bacala.[6] "Our experience of the effect of carbazochrome salicylate on 317 surgical indications and 13 obstetrico-gynecological conditions, has been therapeutically encouraging and successful for the control of capillary bleeding. Foremost among the cases studied were 223 tonsillectomies definitely benefited by this metabolic hemostat, making a diminution of the control incidence of post-tonsillectomy bleeding of 19.8% down to 7%. It has also been found useful in gastro-intestinal bleeding, cataract extraction, epistaxis, incisional seepage, trans-urethral prostatectomy, menometrorrhagias, cervical ooze, antepartum and postpartum bleeding, threatened abortion, and prevention of capillary hemorrhages during hedulin or dicumerol therapy."

Side Effects

All investigators concur that, at recommended dosage levels, Adrenosem Salicylate is free from toxic effects. No cumulative effects attributable to the drug have been reported.

The only side reaction noted has been a transient stinging sensation in the area of injection when Adrenosem Salicylate is used intramuscularly. As one investigator comments: "The brief discomfort which attends the injection of Adrenosem into the gluteal region has not been a significant problem in children or adults as originally anticipated."[5]

Indications

Idiopathic purpura, retinal hemorrhage, familial telangiectasia, epistaxis, hemoptysis, hematuria.

Postoperative bleeding associated with:
tonsillectomy, adenoidectomy and
nasopharynx surgery;
prostatic and bladder surgery;
uterine bleeding;
postpartum hemorrhage;
dental surgery;
chest surgery and chronic pulmonary bleeding.

Dosage

For recommended dosage schedules, please send for detailed literature.

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Tablets: 1 mg. S.C. Orange, bottles of 50.
Tablets: 2.5 mg. S.C. Yellow, bottles of 50.
Syrup: 2.5 mg. per 5 cc. (1 tsp.), 4 ounce bottles.

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8. Fulton, M.D., Dept. of Biology, Boston University: Personal Communication.

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1. Becker, B.: Chlorpromazine–A New Anti-Emetic Agent, Am. J. Ophth. *38*:576 (Oct.) 1954.
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4. Fritz, M. H.: Thorazine® as Preoperative Medication in Ophthalmology and Otolaryngology, Eye, Ear, Nose & Throat Monthly *34*:515 (Aug.) 1955.

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References: 1. Bunim, J. J., et al.: J.A.M.A. 157:311, 1955. 2. Forsham, P. H., et al.: Paper presented at First Internat. Conf. on Prednisone and Prednisolone, New York, May 31-June 1, 1955. 3. Perlman, P. L., and Tolksdorf, S.: Scientific Exhibit presented at A.M.A. Annual Meet., Atlantic City, June 6-11, 1955.

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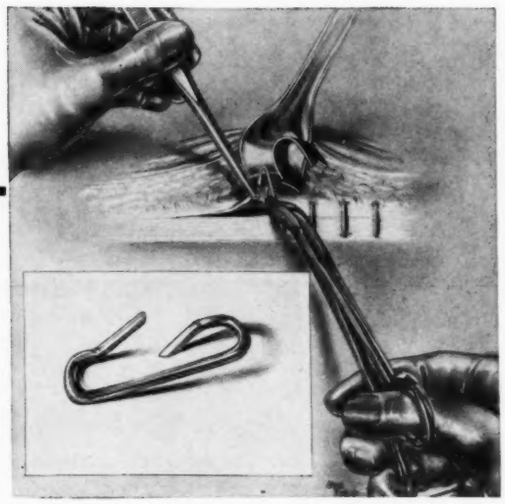

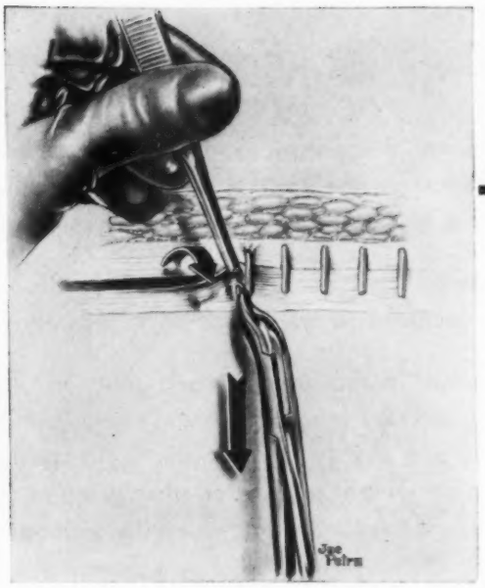

CLOSURE OF ABDOMINAL FASCIA WITH RIGID WIRE—

By Paul I. Hoxworth, M.D.
Cincinnati General Hospital

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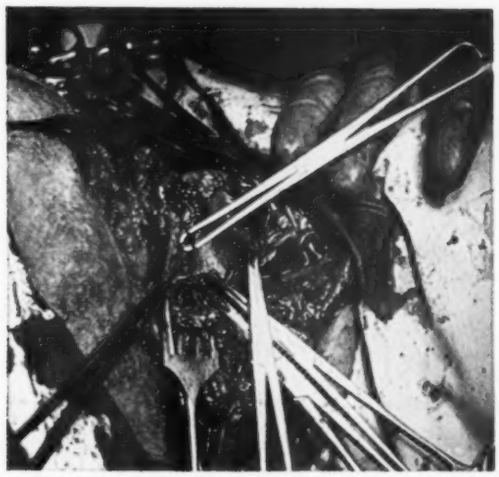
1. Preoperative radiograph of *larynx*. Arrow indicates diseased *epiglottis*.

2. Added incision for *right angle flap*.



3. *Hyoid bone* (right side) about to be removed.

For additional steps in surgery, turn page.

Radiography and Photography

...keys to wider medical knowledge

On this page and the page following, a radiograph and six color photographs show steps in an epiglottic resection.

But, in addition to highlighting the surgery for today's eyes, this visual record does more. It keeps important information "alive" for years to come . . . demonstrates, vividly and memorably, how radiography and photography aid medical knowledge . . . shows how each assists the physician in discussion and teaching, in research and review.

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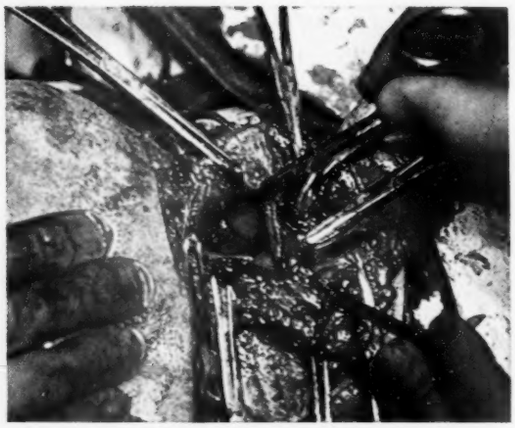
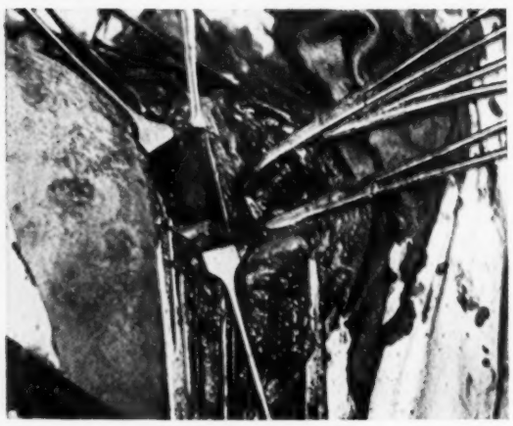
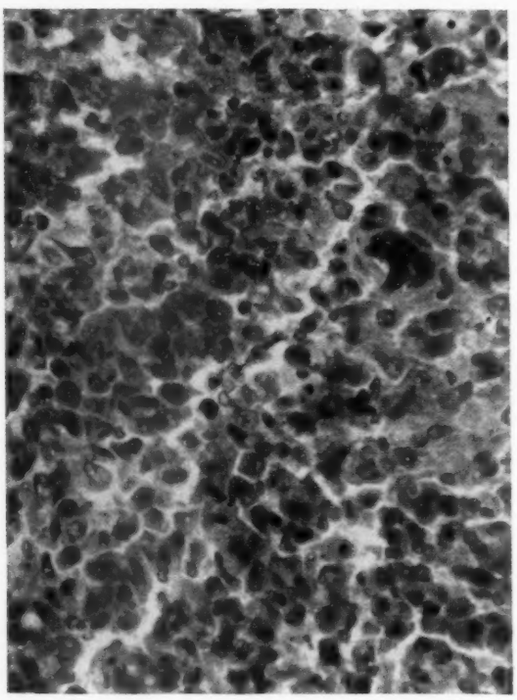
4. Incised portion of *thyroid cartilage* being removed.



5. *Pharynx* opened. Instruments point to *epiglottis.*



6. *Epiglottis* removed. Feeding tube in place.



7. Photomicrograph of *resected epiglottic tissue.* (Magnification, X310.)

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References: 1. Rhoads, J.E.: Internat. Abstr. Surg. *94*:417, 1952. 2. Mecray, P., Jr.: Am. J. Clin. Nutrition *3*:461, 1955. 3. Cannon, P.R.: J.A.M.A. *135*:1043, 1947. 4. Halpern, S.L., in Nutrition in Infections, Miner, R.W., ed.: Ann. New York Acad. Sc. *63*:147, 1955.

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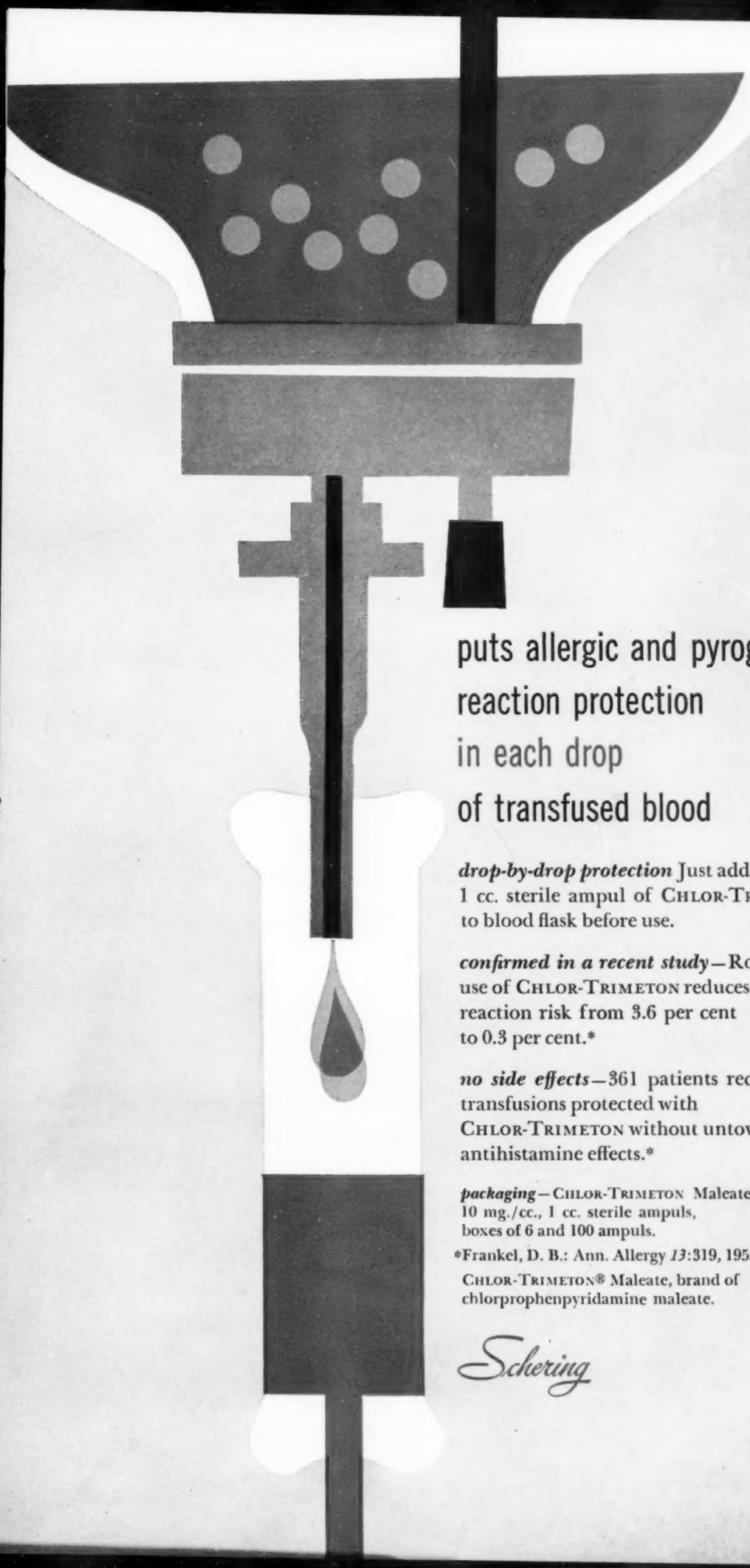

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*Frankel, D. B.: Ann. Allergy *13*:319, 1955.

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1. Hankins, J.R., and Yeager, G.H.: J.A.M.A. *155*:1306 (Aug. 7) 1954.

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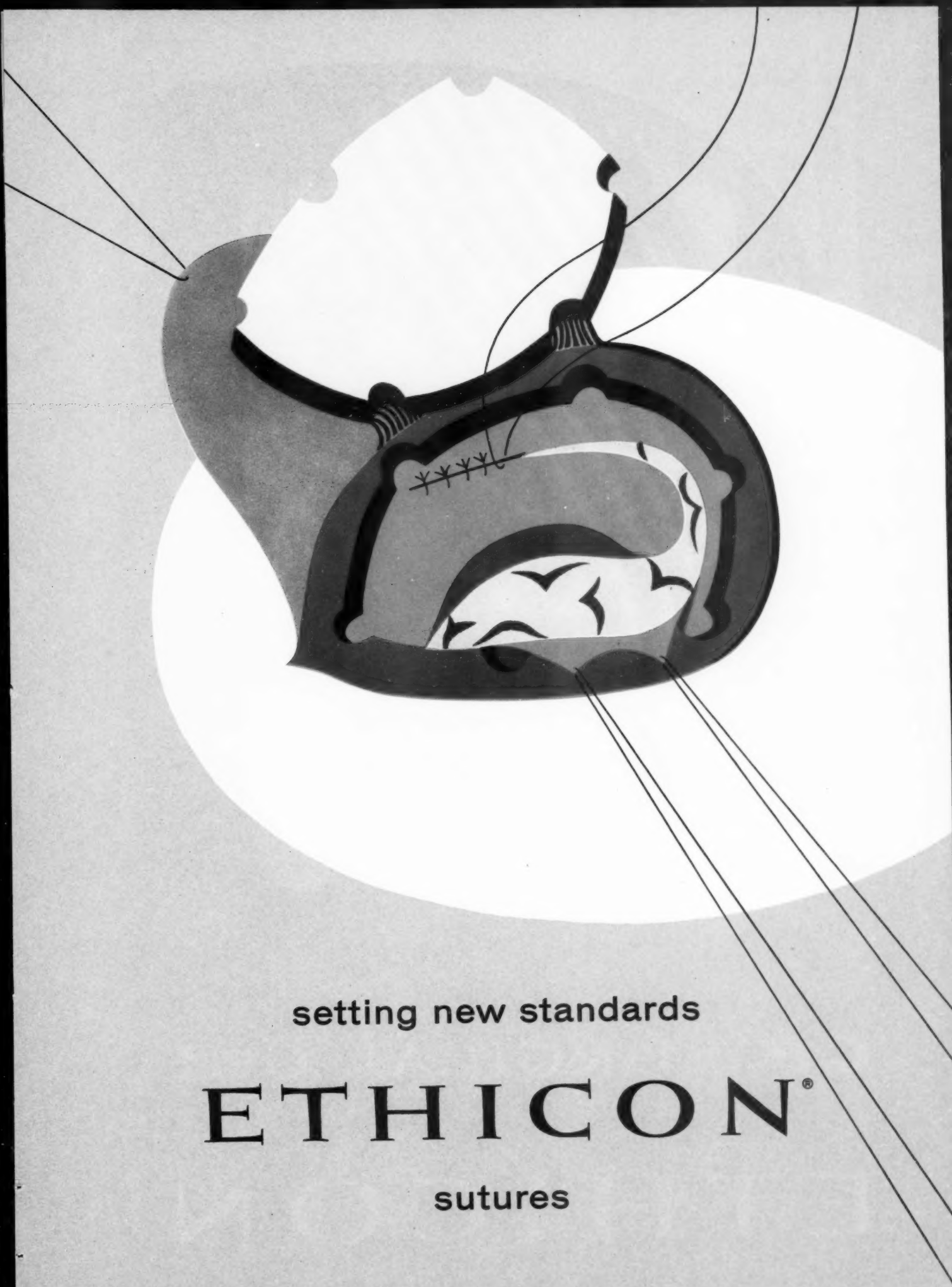
References: 1. J.A.M.A. 153:1516, 1953. 2. M. Clin. North America 27:189, 1943. 3. Surgery 18:200, 1945. 4. N.N.R. 1954, p. 107.



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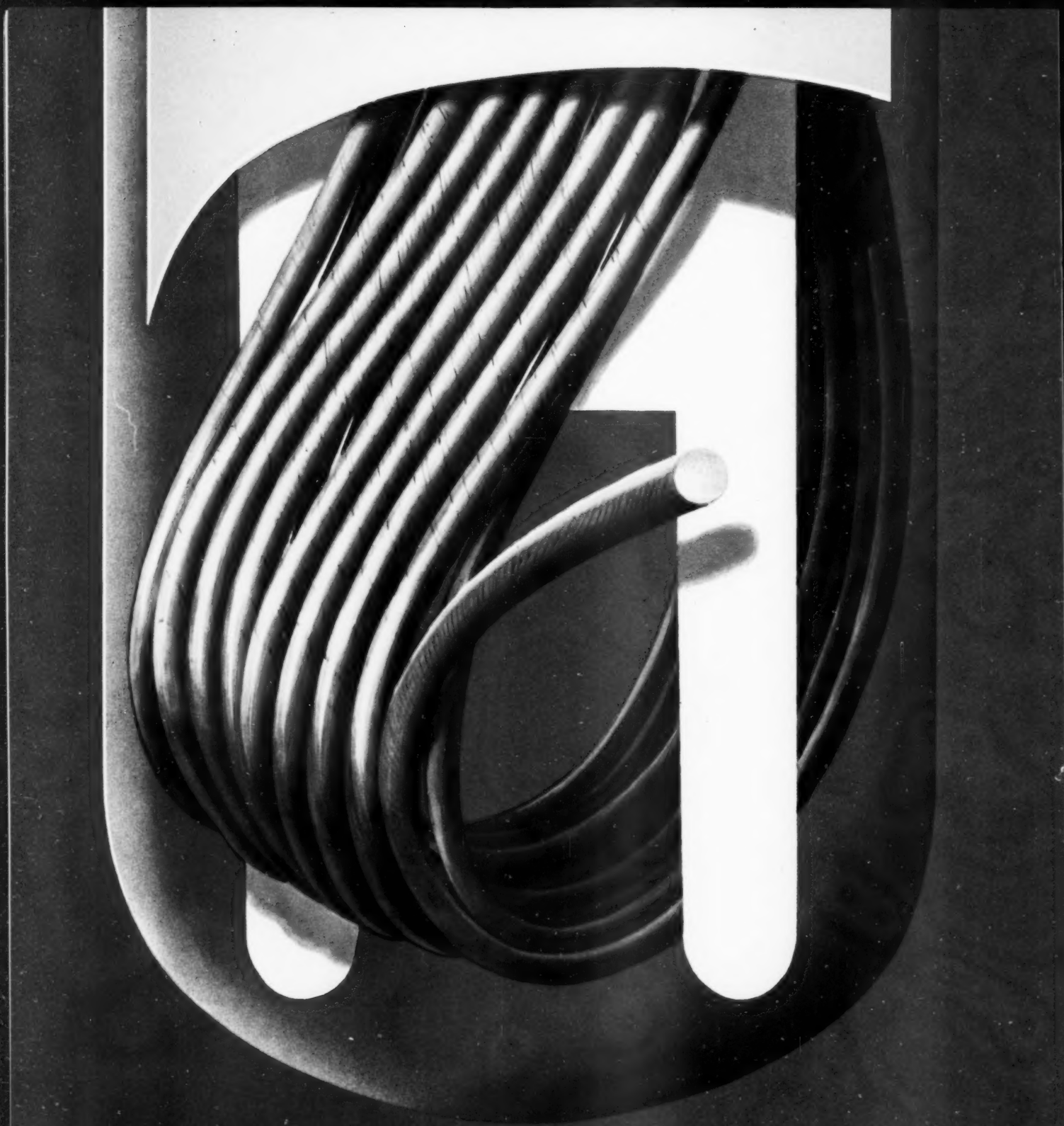

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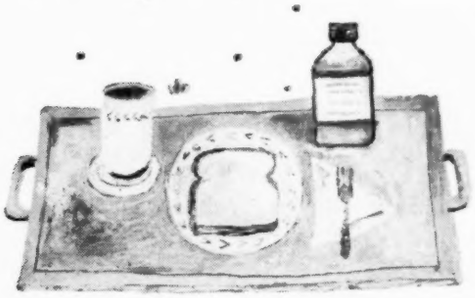

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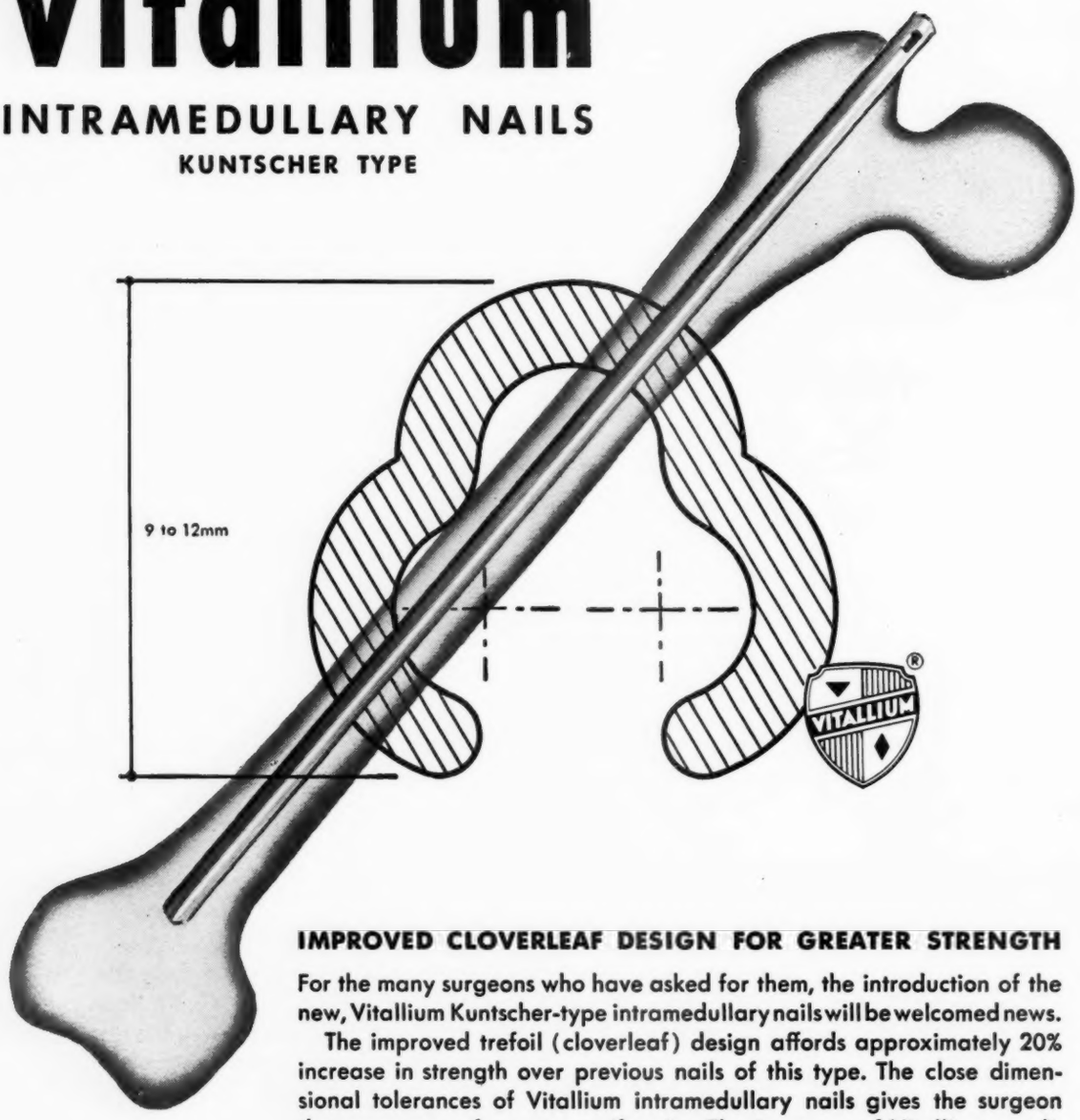

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1. Swanker, W. A.: The use of Clorpactin WCS-90 as an Antiseptic in Surgery. Amer. Jrnl. of Surgery, Vol. 90, July, 1955.
2. Sanders, Murray, & Soret, Manuel G. Virucidal activity of WCS-90. Antibiotics & Chemotherapy, Vol. 5, No. 11, Nov. 1955.
3. Wolinsky, Emanuel, Smith, Marjorie M., & Steenken, William. Tuberculocidal Activity of Clorpactin, The New Chlorine Compound. Antibiotic Medicine, Vol. 1, No. 7, July 1955.

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REFERENCE: Adriani, J., and Kerr, M., *Surgery* **33**:731, May 1953.

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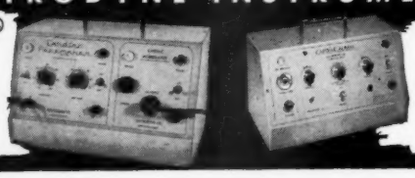

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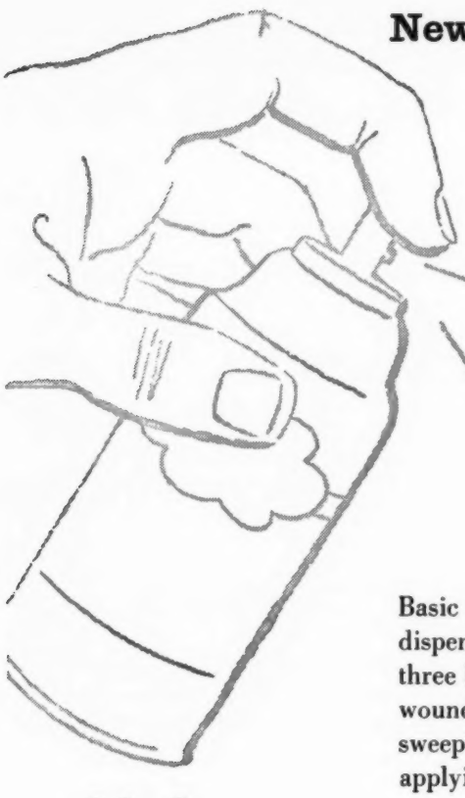

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1. Vibesate (Aeroplast), New and Nonofficial Remedies, 1955, p. 541.
2. Choy, D.S.J.: A.M.A. Arch. Surg. 68:33 (Jan.) 1954.
3. Rigler, S. P., and Adams, W. E.: Surgery 36:792 (Oct.) 1954.
4. Wexler, D. J., Bromberg, F., Rakieten, M. L., and Ryan, B. J.: Am. J. Surg. In Press.

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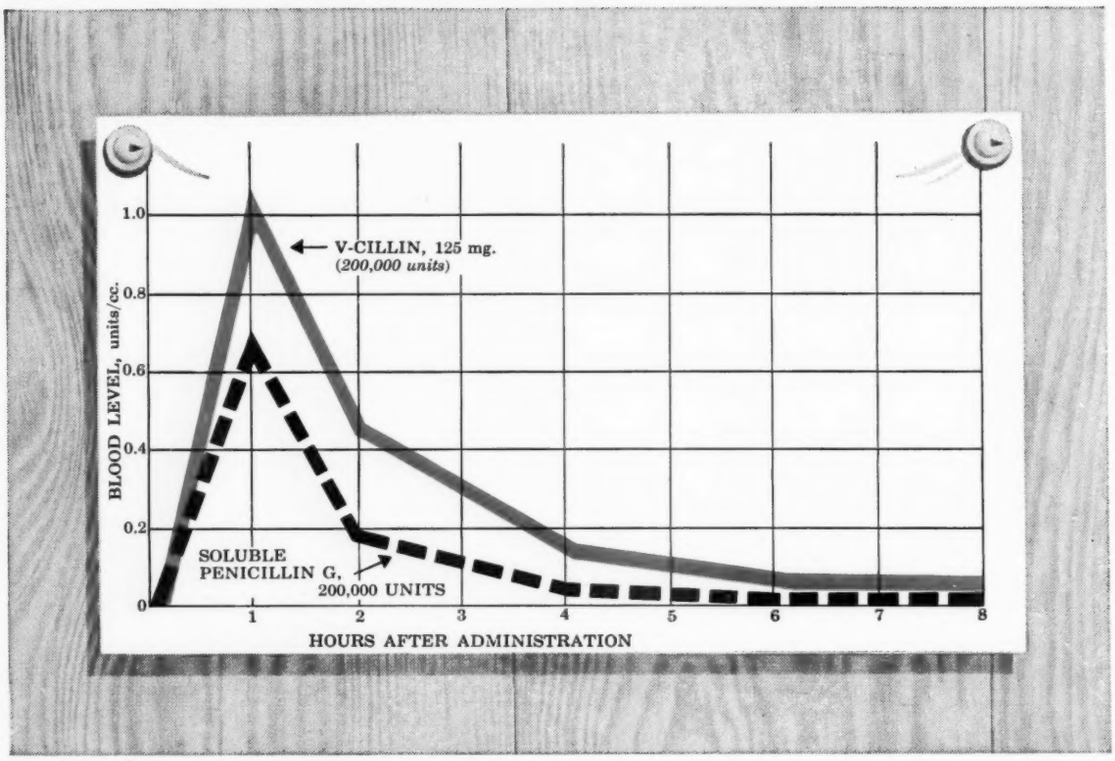

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The American Journal of Surgery

VOL. 91 APRIL 1956 NUMBER FOUR

Presidential Address

PROBLEM AREAS IN THE SURGERY OF TRAUMA

FIRST, I wish to express once more my appreciation of the opportunity to serve as President during the past year. This Association in its seventeen years of existence has taken an honored place among national specialty organizations. The purposes visualized and expressed by its founders have been strikingly demonstrated to be worth while and necessary to progress. We should endeavor constantly to widen our influence in this field. This aim includes many facets.

We have lived through a period in which the scope of surgery has been greatly broadened; in which the patient has benefited by the attack on organs which once were thought to be untouchable, e.g., lungs, liver, pancreas and heart valves. At the same time new methods of treatment have practically abolished the need for surgery in many types of lesions, such as the toxic thyroid, suppurative tenosynovitis, acute hematogenous osteomyelitis, tuberculosis of bones, joints and lymph nodes, empyema, etc. Some day even cancer will be amenable to some treatment other than gross mutilating destruction by surgery or radiation. It is within our imagination that almost all the surgery required at some time in the future will be for congenital anomalies and the results of accidents. We have taken on a field which may include most of surgery within the foreseeable future.

We should be interested in, and working sponsors of, all forms of accident prevention. The National Safety Council, industry, labor, the Armed Forces, insurance companies, the American Red Cross, the Boy Scouts and many other organizations play an active part in this program. We must cooperate and guide each of them both in our home communities and nationally. During the past year real progress has been shown in the field of automobile accident prevention. Impetus for this was furnished largely by the medical profession. Some of the automobile producers insisted that the introduction and publicizing of safety devices would jeopardize their sales and that they would do nothing until demand came from the public. Nevertheless, with 36,000 deaths and 1,250,000 disabling injuries from automobile accidents in 1954 it seemed that the public deserved to have available any known means of protection. Now two of the large companies have installed in their 1956 cars doors that will not be thrown open on impact at 35 miles an hour, steering wheels that do not crush the chest, safety belts and padded panels. Undoubtedly we shall see the other producers follow this wise trend.

The Surgeon General's office of the Department of the Army has instituted a program to demonstrate the handling of mass casualties. This is being given in the larger cities in all parts of the country. We must not consider this as referring to atomic bomb explosions exclusively, but rather of immediate practicality in disasters like the Texas City ship explosion of 1947; the tornadoes of 1953 in Worcester, Flint and Waco; the floods of 1955 in the North Atlantic States. In almost any hospital the arrival of even ten or twenty casualties in a half hour, let us say, constitutes a disaster for that institution. We must be thoroughly prepared to receive mass casualties in every one of our institutions. The members of this society must back this effort.

Legislation, both national and local, appears to be needed in some fields of accident prevention. For example, Dr. Carl Moyer's paper on

burns at our meeting in 1953 brought this home to us forcibly as regards inflammable clothing and open flame heating elements. We must continue to sponsor efforts in these fields.

Education in the field of trauma of many undergraduate medical students is still in a chaotic state. More and more the medical schools must limit their teaching to basic fundamentals rather than give a bird's eye view of the opportunities in each of the many specialties. What can be more fundamental than an understanding of the cause, recognition and care of shock or the intimate details of the pathology, treatment and progress to healing of a wound? These are the bases of all of the care of trauma, but knowledge of them is woefully lacking as most students enter internships and residencies. There should not be a department of trauma in medical schools; trauma cuts across all fields of medicine and surgery. With the amount of time that most doctors will have to devote to problems of trauma when they enter practice, I hope that some schools will install a coordinator in this field such as has been done in the field of cancer under the U. S. Public Health Service. We need not ask for more hours to be assigned for the teaching of trauma, but a coordinator could persuade the professors in charge of each specialty to use some of their regular time at least to touch upon the traumatic problems in that specialty. Injuries are, and will continue to be, among the most common lesions in each specialty requiring treatment.

Only a few medical schools in this country and Canada give any instruction in first aid. Many lay persons have considerable knowledge in this field, and our doctors should be able to guide them and suggest changes in the program as progress occurs. A course comparable to the eighteen-hour standard American Red Cross First Aid Course given to each first year medical student might mean much for his interest in the problems of trauma throughout his medical career; it would allow him during his student days (when he is frequently addressed as "Doctor") to know as much about rendering first aid care as the average layman. Otherwise he will frequently never get this instruction in medical school or afterward.

Internships, and more particularly residencies, have developed into the places for actual practical teaching of treatment. We know that the field of medical knowledge is now too large for this to be obtained in four years of medical school. There are few good surgical residencies where on completion the young surgeon cannot perform a competent subtotal gastrectomy or sigmoidal resection. Usually it will require several years in private practice before he has much, if any, opportunity to put this training to use. In many moderate-sized communities he will be called upon to treat accident cases and it is in this way that he will make most of his living in the first few years. Many residencies allow him little opportunity in this field. Without an ambulance, major traumatic cases will be few. The emergency room is frequently manned by the most junior intern, and supervision and teaching there by senior men is usually lacking. Further, emergency surgery seems to be rarely looked at as having the same glamor as the major interval scheduled case in surgery. Therefore, commonly the chief resident is quite stumped when he is suddenly faced with the problem of a fractured pelvis with possible bladder, urethral or intestinal injury.

It is our duty to see that all surgical residents are exposed to the diagnosis and care of many varieties of trauma, even if this requires rotating them through other services in the same hospital or having them spend a period in another institution. The need for this instruction has been well shown in oral examinations of the American Board of Surgery. For several years all examiners have been asked in writing to stress especially the field of trauma since this has been found to be one of the two weakest fields. The pendulum is swinging slowly toward emphasis on trauma in the residency program, but we must urge its speeding up if our residents and injured persons are to get a "square deal."

While I am not in favor of a chair in trauma in a medical school, I think the idea of a trauma service in a teaching hospital, or one with a residency program, is excellent. Such a service is not easy to institute because of professional personalities and jealousies. There are a few only of these in existence, but once in action under an enthusiastic chief they have usually proved a great boon to everyone. This is especially true for the person with possible multiple injuries, which are becoming more frequent all the time. The individual with a known head injury and nothing else may well be sent to the neurosurgical service; the patient

with a stove-in chest only may go to the thoracic service; the fracture of long bones of the extremities and no possibility of other concomitant injuries may be treated on the orthopedic service. However, the person with two of these, or all combined, belongs on none of these services on admission, but rather on a general surgical service, or trauma service if there is one. One man must be in general charge of the case and he should be one with a general surgical background, not a limited specialty. He should use consultants and definitive care by these specialists unstintingly. Further, when major care becomes necessary in one field only, it should be incumbent on the general surgeon to turn the management of the case over to the specialist concerned.

There are many hospitals in which an x-ray diagnosis of fracture made in the admitting room automatically admits a patient to the orthopedic service no matter what other injuries he may have. I consider this one of the most dangerous directives issued in recent years. I know of hospitals in which an unconscious man is admitted to the orthopedic service because a skull film shows fracture. Is the care of such a patient the aim of usual orthopedic training? I have known of a patient with fractured ribs being sent to the orthopedic service because of hospital orders. How many orthopedic surgeons have been grounded in thoracic physiology and are trained to treat tension pneumothorax or paradoxic respiration? The orthopedic surgeon might well be the first to refuse to go along with a hospital or medical board directive that an x-ray positive for any bone fracture requires treatment of the patient on the orthopedic service. We must lead in clearing up this matter.

Whatever we succeed in doing in medical schools and hospitals to improve education in trauma, the demand from the practicing doctor for further training and keeping up with new ideas will always be terrific. We must foster postgraduate education in our medical schools and at every crossroad. A major part of the treatment of trauma will always be carried out by the general practitioner. I know no meetings which are better attended than those at which injuries are being discussed. These men have to treat injuries all the time, and the results of trauma are often more evident daily to one's neighbors than the results of subtotal gastrectomy or colectomy. The proper immediate treatment of a hand injury will usually be more important to the individual for the rest of his life than how well his appendix is removed, a fact which is appreciated by many general practitioners and surgeons in small communities soon after they enter practice. They want help and the members of this society should try to see that they get it. Trauma should appear on the program of every general medical meeting. Our annual gathering is remarkable in the quality of its papers, but it must not be an ivory tower. Each one of us should be a pioneer in fostering postgraduate education for both the surgeon and the general practitioner.

Compensation insurance has created new problems in the field of trauma for over forty years. One of its main purposes was to make it unnecessary for an injured employee to bring suit for recovery, negligence first having to be proved. My own state is one of many in which the attorney has again made himself almost a necessity. I am told that study of the handling of compensation cases has recently been made one of the courses in some law schools. The expense to industry of complying with the compensation law has become excessive. The backlog of hearings often entails many months of compensation payment beyond real disability. The amount of medical payment is a large figure in total hospital and doctors' income, in my own state amounting to over $40,000,000 a year. Many doctors decline to treat compensation patients, whereas for others it is big business. All states vary in their laws, so that the problem in each is different. However, we must admit that much of the medical care is indifferent and unduly prolonged, that union rules seem to prevent a man from returning to any labor except eight hours at his usual job no matter what the desire of the employer is, that the expense of compensation insurance based on experience is too great a charge on industry and on the cost of living, and that final settlement frequently takes too long.

We have heard so much about rehabilitation since World War II that even the word is almost an anathema to many doctors; yet it has a large place in our economy. We believe that the injured person wishes to return to work. The malingerer, at least in the early years following injury, is an extremely rare person. Immediately after injury we are not dealing with a sick man. He is as well as he was before the accident

except for his injury. It is our duty to see to it that he does not become a sick man while under our care. We, as individual doctors, are responsible for the injured man from the moment he is injured until he is back at work, if not in the same job at least in the best position for which he is physically and mentally fitted. This means we must take the lead in first aid care in our communities, in the training and competence of ambulance personnel, in the manning and supervision of emergency rooms, in the prevention of referral to incompetent doctors by ambulance chasers. Once the patient is in an approved hospital, the care of the injured part is usually good; but there are too many pitfalls in getting the patient there. Further, too often no thought is given to what will happen after the patient leaves the hospital. All parts of his body not actually injured or immobilized should be exercised at least once a day from the day of injury, on orders of the doctor. This well patient must not be allowed to deteriorate before our eyes and then require at least a similar amount of time to return to normal. He is not going to exercise unless we tell him to and arrange for supervision and encouragement of this exercise. A healed wound or a united fracture does not mean that treatment is completed. What good are these results if we do not follow the individual through until he is working again, earning a living wage and supporting his family? Otherwise we are merely the unwitting means of adding one more to the 250,000 persons a year who become permanently disabled and charges of the public.

All of us have seen in our own communities a patient who has been furnished with crutches, a back brace or a prosthesis following amputation, and is then discharged from the hospital and promptly forgotten. He has not been told what the next move is or whether he will ever return to his job. Often his attorney becomes the patient's best friend, apparently. He will not allow the crutches or brace to be discarded until the case is settled. Meantime the patient's local and general musculature and morale are steadily deteriorating, complaints increase, new symptoms develop and the tag "functional" or "neurotic" is fastened on him. Soon we are fortunate if this well person, now completely recovered from an accident to one part, is ever able to return to the labor market. We must accept at least equal responsibility with the man himself for this tragedy. We did not exert ourselves to keep a well man from getting sick under our eyes. The care of a wound, the "cabinet maker's work" on a bone, may have been excellent, but the man was not made to feel that he was as good as he ever was and that his take home wage would again be assured. Surgery is an art, and the field of the surgery of trauma cannot be practiced in the operating room and routine rounds only, or even mostly.

The lump sum settlement is one of the most unfortunate features in the compensation laws of many states. Hearing after hearing, one postponement after another, exposure to testimony until the claimant knows all the answers, arguments among doctors, advice against work until the permanent disability is decided upon and finally a lump sum settlement is made—three, five, seven years after the accident. Rarely can rehabilitation be adequately effective after that length of time and frequently it has not been carried on at all, or seriously, before final settlement is made. There appears to be fear that rehabilitation may reduce the amount of the settlement. In 1951 among 4,430 industrial patients returned to work after referral for vocational rehabilitation under the Federal-State program, the average time lag since injury before referral was seven years. Rehabilitation must start on the day of injury and continue throughout convalescence until such time as the injured person has made the most complete recovery possible and is back earning wages. The laws should be framed so that up to that time the injured person and his family can live as human beings and no lump sum settlement is made for permanent disability until he is working, if this will ever be possible. We should each play our part in obtaining better compensation laws which will promote rehabilitation.

These are some of the problem areas in the surgery of trauma. None of them allows a permanent solution. Each one is subject to evolutionary change with new conditions. As this Association infuses fresh blood into its membership each year, we should expect that it will be kept from becoming hidebound, sterile and unproductive. The opportunity is tremendous. Here are dreams and the fulfillment of dreams for all of us.

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Current Therapy of Tetanus

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TETANUS is a disease known to antiquity. It has usually been considered of importance only in relation to war wounds where in the past its incidence was 2 to 16 cases for each 1,000 wounds. However, it has sometimes occurred in considerable frequency in civil practice, as in tetanus of the newborn which in the 18th and 19th centuries was a common disease reaching such proportions on occasion that in the Rotunda Hospital, Dublin, from 1757–1782, 16 per cent of all newborn infants died of tetanus.[1] Furthermore, tetanus may occur in great frequency in civil disaster. Glenn[2] has described the high incidence of tetanus in civilians wounded in the siege of Manila, and we might anticipate such problems in the future in wholesale wounds occurring in a largely non-immune civil population exposed to modern day super bombs.

The relatively low incidence of tetanus in the United States today is undoubtedly influenced by the widespread use of prophylactic antiserum in civilian injuries; still, 500 to 600 deaths each year are attributed to that disease in this country.[3]

Fortunately, the mortality of tetanus has been improved by modern treatment and has fallen from 80 to 100 per cent[1] to the neighborhood of 35 per cent.[4,5] Perhaps these figures will be further improved.

Tetanus in our community is rare, as it is in most northern states. In the past seven years we have treated five cases, and on the basis of our experiences with these cases we would like to outline what we believe to be a satisfactory program of management.

Diagnosis. The diagnosis is a clinical one and is based on the usual history of a recent injury, often trivial, with the characteristic clinical picture. This consists first of painful rigidity of the muscles of mastication and inability to open the mouth with hyperactive reflexes, followed soon by spasm of the neck muscles and later by more or less of the following: spasm of the back and abdominal muscles and muscles of the extremities, and generalized convulsive seizures. The sensorium remains clear except in periods of hypoxia due to convulsions.

There are very few things to be confused with tetanus even in its earliest stages; and when the diagnosis is suspected, treatment should be started at once if it is to be of any benefit.

Specific Antitoxin. The patient should be skin-tested to the antiserum and then after desensitization, if necessary, given 40,000 units intramuscularly and 40,000 units intravenously. An additional 10,000 to 20,000 units should be injected locally about the wound before any operative manipulation. Subsequently intramuscular antitoxin should be given in 10,000 to 20,000 unit daily doses for five to seven days. This schedule of dosages is somewhat arbitrary and may be excessive but is done in the interest of conservatism.[1,3–5]

Wound. The wound should be excised or débridement done, depending on the local circumstances.

Tracheostomy. When there is severe spasm of the muscles about the face, throat and neck, the patient has difficulty in swallowing and in coughing and clearing secretions from his respiratory passages. This interferes with respiration, promotes pulmonary complications and during the resultant choking episodes stimulates generalized seizures. Therefore tracheostomy should be a planned step in therapy of any patient with marked spasm of muscles about the face and throat, and preferably should be performed when the wound is treated or at least before generalized convulsions begin.

Sedation. We have used barbiturates as well as chloral hydrate and paraldehyde effectively. Sedatives must be used in large doses during the stage of convulsions, and we have used as much as 6 gr. of sodium luminal® every four hours intramuscularly along with 1 ounce of paraldehyde every six hours rectally. The objective is to keep the patient dozing lightly without reaching the stage of respiratory depression. As soon as the muscle spasm begins to diminish, the dosage of sedatives must be reduced sharply. Opiates may be used to help relieve pain from muscle spasm.

Muscle Relaxants. Much has been written about the use of curariform drugs in the treatment of tetanus.[4–9] The most radical proposal is that of Lassen and associates[7] who recommend complete curarization and positive pressure-controlled respiration as long as necessary, in their hands as long as seventeen days. Workers who have used these drugs on large numbers of patients are not enthusiastic.[4,10] It has been pointed out that the rigidity and convulsions *per se* do not affect the outcome of the disease[4] and, moreover, curare will not prevent the convulsions unless carried to the point of respiratory paralysis. This latter state multiplies the problems of care. We have used curare-in-oil to help reduce the rigidity with the belief that it might alleviate pain somewhat; however, the result was variable. We have relied on deep sedation to prevent the stimulation of convulsions. In one case during the worst stage general anesthesia with sodium pentothal,® nitrous oxide and oxygen and curare was twice induced for periods of about one hour each. Subsequently the patient was much more relaxed for eighteen to twenty-four hours.

General Care. The patient should be placed in a quiet, darkened room and protected from stimulation as much as possible. The utmost in nursing care and observation is essential, with a minimum of manipulation of the patient. Tracheal secretions are removed by suction quickly and efficiently only as often as needed. An indwelling catheter is essential in most patients with severe tetanus.

The patient may be given liquids through an inlying gastric tube of small caliber polyethylene.

Antibiotics are used primarily to combat respiratory infection.

In our series of five cases all had incubation periods of twelve days or under and all could be classified as severe with generalized rigidity and convulsions for five to ten days. Three occurred after head wounds. One death occurred in an eighty-one year old man in the third week of his disease when the spasm had begun to recede. Death was due to urinary tract infection, uremia and bronchopneumonia, and must be considered to have been preventable.

Prophylaxis. Few of us dare treat wounds of any size or any degree of contamination in an unimmunized individual without giving tetanus antitoxin. Such passive immunization with 1,500 to 3,000 units of antitoxin lasts for ten to fourteen days and in all probability reduces the risk of tetanus. However, the incidence of reactions is considerable. Serum sickness alone develops in about 10 per cent of individuals given horse serum, and this may be disabling and severe. Furthermore, as Stafford[11] has pointed out, in sensitive individuals, whether or not subjected to a course of desensitizing injections, the foreign serum will be destroyed and excreted much more rapidly, probably in four or five days largely eliminating any potential benefit from passive immunity. We would agree with Stafford that antitoxin in the individual sensitized to the vehicle serum should not be used prophylactically. Since the work of Ramon[12] we have a highly effective means of producing active immunity by means of tetanus toxoid. During World Ward II tetanus was almost eliminated from the American Armed Forces. The Army reported only twelve cases of tetanus in 2,734,819 wounds or injuries, and only four of these had had the prescribed course of toxoid immunization and booster.[13]

We are now finding that the immunity established in service personnel ten and more years ago is still effective.[14–16,23] Stafford, Peterson and their associates have shown that in groups tested five to eleven years after the last booster injection of toxoid, over half still had an effective level of circulating antibiotics. Furthermore, all showed an accelerated response to a booster dose of toxoid with sharp rises in circulating antibodies in four to seven days.[15,16,26] It would seem that this information coupled with the realization that the incidence of reactions to toxoid is extremely low[13,17,18]

(sensitivity reactions to toxoid in the U. S. Army fell to below 2/100,000 injections by the end of World War II[13]) makes a strong case for the promotion of active immunization programs among the adult population. I think this should particularly appeal to industries where danger of injury is relatively high. This has already been reported by at least one industrial organization.[19]

Furthermore, we should recommend booster injections of toxoid be given to all previously immunized persons not more than five years apart until sufficient time has elapsed that further studies may more accurately establish the safe interval.

A serious question arises concerning the unimmunized person given prophylactic antitoxin at the time of injury: Should he simultaneously be given a first dose of toxoid? Considerable work has been done on this problem and numerous authorities have expressed opinions that antitoxin and toxoid can and should be given at the same time.[3,11,16,20,21,24] However, the weight of published experimental evidence would seem to indicate that in the individual not previously immunized the primary response to tetanus toxoid is inhibited by a higher titer of circulating passive antibodies.[20,22–25] This appears to be true even when repeated daily doses of toxoid are given and is in contrast to the effect of simultaneous antitoxin and toxoid in the previously immunized individual where the secondary accelerated response to the booster is not greatly affected.[24] Conversely, however, there does not seem to be any significant interference with the effect of passive antitoxin by simultaneously given toxoid.[23,24] Nevertheless it seems to be more in keeping with experimental evidence to wait for two to three weeks after passive immunization before starting a course of active immunization with toxoid.

Furthermore, toxoid immunization should be carried out after recovery from clinical tetanus, as the latter has been demonstrated to excite no lasting immunity.[23]

SUMMARY

We have outlined a course of management for tetanus based on experience with five cases.

Recommendation is made relative to active immunization against tetanus.

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Delayed Surgical Infections Following Antibiotic Therapy

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WITH Domagk's report of the effectiveness of prontosil® against hemolytic streptococcal infections[1] in 1935, a new era in therapeutics for bacterial infections began. This was followed shortly with the discovery by Trefouel et al.[2] of sulfanilamide, as the active principle of prontosil. During the next decade numerous allied chemotherapeutic agents were developed.

Sir Alexander Fleming predicted early that the sulfonamides would not fulfil all the requirements for the control of bacterial infection. As a result intensive investigative work was conducted along a path which led to the eventual production by Chain et al.[3] of sufficient penicillin to demonstrate its action *in vivo*. It was soon evident that penicillin had its limitations and was not a panacea. Through constant research a variety of new antibiotics were produced which were effective against penicillin-resistant bacterial organisms.

During the past two decades the medical profession has been deluged with a variety of antimicrobial agents. Many have been and are of immeasurable value when properly used; others for one reason or another have been short lived. The use of many of these preparations by the medical profession has become so commonplace that a large portion of the population has at one time or another been treated with one or more antimicrobial agents.

Penicillin is now being manufactured at a rate well over 300 tons a year, streptomycin is being produced at over 100 tons a year and the broad-spectrum antibiotics are being manufactured at over 250 tons a year. The usage of such overwhelming quantities of these agents is having a profound effect upon bacterial infections in man.

In a discussion of the effects of antibiotics on the host, conducted by the Royal Society of Medicine, the following effects were discussed: (1) toxicity, (2) sensitization, (3) vitamin deprivation, (4) super-infection and (5) growth promotion.

Several of these effects have become so well recognized that they have been termed "antibiogenic syndromes."[5] The current medical literature contains a myriad of reports on many phases of the undesirable side effects of antibiotics, however, we have found only a few brief references[5–7] to delayed surgical infections. It is our belief that delayed surgical infection is a distinct undesirable side effect following the use of antibiotics. We further believe, particularly from a surgeon's point of view, that this phenomenon deserves separate consideration. Thus we were prompted to study the problem further.

In the past few years it has become evident to us in studying cases of delayed wound infections that a typical pattern has been produced when antibiotics were used prophylactically. This pattern is best illustrated by the following two case reports:

Patient J. R., a forty-eight year old white man, was admitted to the hospital for gastric resection for intractable duodenal ulcer. The operation was performed with minimal contamination. The patient received combiotic® twice daily, prophylactically. His postoperative course was uneventful; the drainage tube was shortened on the seventh and eighth day and removed on the ninth day. He was discharged from the hospital on the twelfth day with his wound well healed. The highest temperature occurred on the second and third postoperative days but never went above 100.2°F. On discharge from the hospital, the patient was afebrile. On the eighteenth postoperative day his temperature rose to 103°F. and on the following day the operative wound became tender and painful. Upon examination, the entire wound was red and fluctuating. A localization of 150 cc. of frank purulent material was evacuated. A drainage tube was placed in the wound. Further convalescence was uneventful. The culture showed coagulase-positive Staphylococcus aureus.

Patient S. J., a one-day old female infant, was found to have a mass herniating into the cord at the time of delivery (normal, spontaneous). A diagnosis of omphalocele was made. This was repaired the same day and the infant received penicillin and streptomycin, prophylactically. The postoperative course was uneventful. The infant was afebrile at the time of discharge from the hospital nine days later. The wound was well healed at this time. On the fifty-eighth postoperative day the baby's temperature rose to 104°F. and the following day a mass developed at the site of the previously repaired omphalocele. The infant was readmitted to the hospital and incision and drainage were performed, evacuating 75 cc. of purulent material. Culture showed coagulase-positive Staph. aureus and gram-negative coli of the Escherichia coli group. Sensitivity studies showed that the antibiotics used prophylactically were ineffective against the organisms cultured.

To date we have collected sixteen cases of delayed postsurgical infection. All cases were instances in which antibiotics were used prophylactically since at the time of surgery there was no acute infectious process or suppuration present. Fourteen patients received penicillin or penicillin dihydrostreptomycin combinations postoperatively. Two patients were given broad-spectrum antibiotics. At the time of discharge from the hospital all of the patients were afebrile and there was no evidence of wound infection at the time. In each case, a sudden temperature rise developed, followed by the appearance of an infectious process. The infection appeared between the sixteenth and twenty-fourth day postoperatively, with an average of seventeen days. Cultures were obtained in thirteen of the sixteen cases. In three instances the cultures showed gram-negative coli of the Esch. coli group and in nine cases the culture showed coagulase-positive Staph. aureus. In the cases in which sensitivity studies were conducted, none of the organisms cultured were sensitive to the antibiotics that had been used prophylactically.

COMMENTS

As is illustrated by the cases in this series, a common pattern is evident in that prophylactic antibiotics were used in apparently clean operative cases. An uneventful hospital course followed. However, when the patients were discharged from the hospital and antibiotics were discontinued, a sudden rise in temperature developed along with a suppurative process intraperitoneally or within the incision. After the institution of surgical drainage, all patients convalesced uneventfully. Analysis of the sensitivity studies which were conducted in ten of the thirteen cases in which cultures were obtained, showed that in not a single instance was the organism cultured sensitive to the antibiotic administered prophylactically. The recent literature has shown a progressive increase in the yield of penicillin-resistant strains of coagulase-positive Staph. aureus from 14 per cent in 1946[8] to 75.8 per cent in 1953.[7] It has also been shown recently that there has been a gradual emergence of resistant strains of bacteria to aureomycin and chloramphenicol.[9] Howe[7] has pointed out that the emergence of resistant strains is in direct proportion to the quantities of the antibiotic which is used within an institution.

We are in agreement with Meleney,[10] and Altemeier and Culbertson[6] that the indiscriminate use of antibiotics on a prophylactic basis is unjustified. Every effort should be made to establish the use of antibiotics on a rational basis. We believe that this necessitates obtaining cultures and sensitivity studies wherever possible, as has been so often emphasized by Meleney.

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Traumatic Fat Necrosis

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AT the present time, fat is certainly the most unpopular of the tissues and it is said that obesity predisposes to almost every human ill. Increased fat in the blood stream is blamed for many acute and chronic vascular diseases. Fat embolism following fractures is a well known phenomenon. Fat is notoriously susceptible to infection.

In this presentation we would like to point out the vulnerability of fatty tissue to trauma. Although this vulnerability has been recognized for many years, it has not been sufficiently emphasized, in our opinion. Traumatic fat necrosis has never been formally discussed before this society, although Greene and Howard[1] related trauma to hypertrophy of the male breast in 1952. It seems pertinent, therefore, to present a review of the cases of fat necrosis that have been seen at the Hospital of the University of Pennsylvania during the twelve-year period from July, 1942 to July 1, 1954, excluding those cases that were associated with pancreatitis.

The subject of fat necrosis has been discussed in European literature at intervals for nearly 100 years but it was not a commonly recognized entity in this country until 1920 when Lee and Adair[2] described their first two cases of fat necrosis of the breast. In both of their cases fat necrosis followed trauma, and Lee and Adair introduced the term "traumatic fat necrosis" to American literature although this term had been used many years previously.[3] By 1924 Lee and Adair[4] had collected a few additional cases of their own and had gathered examples from the literature, making a total of twenty cases which involved the breast. It is of interest that three of these followed the use of hypodermoclysis in the breast, and fourteen patients or 70 per cent had a definite history of trauma. In the meantime Farr[5] had described seven cases of fat necrosis located in other parts of the body. Two of his patients had fat necrosis after hypodermic injection, and one from trauma at birth. Farr referred to the lesion as "ischemic fat necrosis."

Subsequently the lesion has been recognized with increasing frequency throughout the world; and although it was once considered to be extremely rare, it is now reported frequently. Several papers[6-10] have described this condition in the newborn, usually following a difficult delivery. Fox[11] in 1933 described five cases among the newborn. The babies were all large, and three of the five were firstborn—a combination leading to more than the usual trauma at birth. In the newborn this lesion seems to be self-limiting and within three or four months it has usually resolved completely. Although the authors usually speak of trauma precipitating the trouble in young children, there are infants in whom microscopically similar lesions develop without a history of trauma. Ivy and Howard[12] found the lesion after caesarean section. Fox took pains to differentiate these lesions from other lipoid disturbances that may occur in the newborn such as scleroderma, and sclerema edematosum or Weber-Christian syndrome.

Lesions have been reported in nearly every area of the body where fat is normally found. Hinman and Johnson[13] reported three cases with scrotal involvement, and Galbraith and Young[14] recently reported another. The majority of reports, however, deal with the breast, where the lesion is found most frequently. In nearly every paper dealing with fat necrosis of the breast the authors have emphasized the clinical similarity that may exist between this lesion and carcinoma. In one large series[15] one-fourth of the cases reported in the breast had been treated with radical mastectomy because of the mistaken diagnosis of cancer. This is an error that should not be made. Anyone familiar with the characteristics of fat necrosis should

diagnose or at least strongly suspect it on gross examination at the operating table even if frozen sections are not available.

Etiology. The true cause of fat necrosis is still not known. That recognizable trauma does not always exist prior to its development is well known. In most series reported[15–18] about half the patients have a history of recognizable trauma preceding the development of the lesion. Trauma, when it exists, takes many forms. It may be a direct blow, a hypodermic injection, strangulation of tissue, surgical trauma, radiation or even cold. The occurrence of fat necrosis after cold is interesting and has been reported on several occasions. It was described in the last century and "rediscovered" later in this century. In 1923 Cohen[19] reported a case following the use of an ice pack, and recently Collins et al.[20] described a case that developed after hypothermia used for a cardiac operation on a young child. Harrison[7] pointed out that an infant's fat has a higher solidifying point than an adult's because of its relatively greater palmitic acid and smaller oleic acid content. As a consequence, solidification and subsequent fat cell injury occur more readily in the child, a fact that may be of importance in determining etiology.

In addition, a relationship has often been seen between fat necrosis and infection. One can picture the process starting after some cell destruction incident to the infection. The interference with blood supply may also be a means by which infection may render this fat susceptible to necrosis, and such is the presumed cause in cases following strangulation. Hypoxia alone is also thought to be a causative factor and there is some evidence that this may play a part particularly in those cases involving the newborn.[8]

In all theories of cause one meets the problem of why this lesion develops in only a small proportion of the people sustaining trauma. It seems that some individuals must be more susceptible than others. Consider the boxer or football player and how infrequently he develops fat necrosis. It has been postulated[15] that an increase in blood lipase is responsible for the lesion, but so far as we are aware this theory has not been proved except in cases associated with acute pancreatitis. We are then forced to admit there are many cases which do not fit into the usually postulated causes and for which we have no good explanation. There is little doubt, however, that in a considerable proportion of cases trauma seems to be a precipitating factor.

Pathology. On gross section, an area of fat necrosis is recognizable as a light yellow or white opaque area that may have some liquid fat in its center. The lesion does not appear infiltrative but may give external signs of fixation. Its opacity when compared to normal fat is usually striking; microscopically, the normally clear appearance of the fat cells is lost in the early stages of necrosis and instead they take on a cloudy appearance. Boyd[21] states this is due to saponification rather than true necrosis. Crystals of fatty acid may also be present and eventually combine with calcium ions. Later, necrosis is seen with disruption of cell membranes, and around the necrosis newly formed connective tissue develops. Giant cells are often present as are inflammatory cells, and obliteration of the smaller blood vessels is frequently noticed. The period of time required for a firm, clinically recognizable lesion to appear after trauma is variable. It usually takes several weeks but may develop in from one week to many months. It is our belief that if surgical wounds were routinely examined microscopically a week or two after they were made, many of them would show areas of fat necrosis that subsequently resolve. Our experience with wounds of the breast, described herein, lends support to this belief. In the cases that do not resolve themselves, however, calcification and eventual cyst formation may occur.

Analysis of Data. Between July, 1942 and July, 1954, at the Hospital of the University of Pennsylvania, there were 163 patients with fat necrosis proved by microscopic examination, excluding all cases associated with pancreatitis. Of these 163 patients, seventy-four had the lesions in their breast, sixty-four in or about the peritoneal cavity and twenty-five in subcutaneous tissue elsewhere than the breast. Of the entire group, 50 per cent were associated with previous trauma, 10 per cent with incarceration or strangulation of tissue and 14 per cent with infection, leaving 26 per cent not related to these main categories.

The apparent causes of the seventy-four lesions found in the breast are listed in Table 1.

The two cases following needle puncture resulted from attempts to aspirate a cyst. The

twenty-six cases caused by surgical trauma followed previous surgery of the breast; six of these patients, having had a carcinoma removed locally a short time previously, were subjected to radical mastectomy, and fat

TABLE I
CAUSES OF FAT NECROSIS OF THE BREAST

Apparent Cause	No. of Cases	Percentage	
Trauma, direct	17	23	61
Trauma, surgical	26	35	
Trauma, needle aspiration	2	3	
Infected cyst or abscess	10	13	
Unknown	19	26	

necrosis was found near the surgical wound. The time interval in these cases varied from five days to six weeks. This finding suggests that many unrecognized cases of fat necrosis occur after surgical wounds and are self-limiting. In none of the seventy-four cases was radical mastectomy carried out for fat necrosis in spite of the fact that many showed clinical signs suggestive of carcinoma, such as skin retraction and the presence of a hard, ill defined mass.

Of the sixty-four instances of fat necrosis noted in or about the peritoneal cavity, twenty-three were found in the omentum, sixteen in the peritoneum or preperitoneal fat, fourteen in the mesentery of the bowel and the remaining eleven in various areas such as the gallbladder, epiploic appendages and mesosalpinx. For

TABLE II
INTRA-ABDOMINAL FAT NECROSIS (EXCLUSIVE OF PANCREATITIS)

Apparent Cause	No. of Cases	Percentage		
Trauma, surgical	21	32	35	58
Trauma, direct	2	3		
Strangulation or torsion	15	23		
Infection	15	23		
Cause unknown	11	19		

purposes of simplification, we have grouped these cases together and Table II shows their apparent cause.

If we group the cases of torsion and strangulation with those cases that are clearly traumatic in origin, we find 58 per cent of our abdominal cases fall in the category of traumatic origin. In none of the cases in this abdominal group was the area of fat necrosis the presenting symptoms or sign for which the

TABLE III
CAUSES OF SUBCUTANEOUS FAT NECROSIS

Apparent Cause	No. of Cases	Percentage	
Trauma, direct	5	20	56
Trauma, surgical	4	16	
Injections, hypodermic	5	20	
Associated with hernias	2	8	
X-ray therapy	1	4	
Infection	1	4	
Degeneration in lipoma	5	20	
Unknown	2	8	

operation was carried out although this has occurred in cases reported by others.[14,22]

The remaining twenty-five cases of fat necrosis involved the subcutaneous tissues exclusive of the breast and have been of great interest to us. They were scattered widely over the body and resulted from quite variable apparent causes as shown in Table III.

Here again we find trauma responsible in over one-half of the cases. The lesion attributed to radiation was in the abdominal wall of a patient who had received radiation for carcinoma of the cervix nine months previously, with a portal directly over the area of the fat necrosis. The relationship may be coincidental but seemed suggestive.

Another interesting instance was that of a newborn baby in whom fat necrosis developed in the preauricular region where forceps had been applied at delivery.

The finding of fat necrosis within a lipoma may well be related to the rather poor blood supply of lipomas.

The predominance of fat necrosis in women is apparent from our series, as is commonly reported. Fat necrosis in the male breast, although reported,[23] is very rare and we did not see it. Of the cases occurring within the abdomen, nineteen of sixty-four (30 per cent) were in men, and in the cases of subcutaneous fat necrosis eight of twenty-five (32 per cent) were in men. Of the entire series, there were only twenty-seven men (16 per cent).

COMMENTS

It is our belief that fat necrosis is a common lesion and is often overlooked. The fact that 163 such lesions were excised within twelve years in our hospital clearly shows that fat necrosis is not rare. In many instances it resolves itself, particularly in young children. It is an entity, however, of which surgeons must be cognizant if they are to avoid some serious errors. It is difficult today to condone amputation of the breast for this benign lesion, for the well trained surgeon should be aware that fat necrosis of the breast can closely simulate cancer on preoperative examination. It is our impression that many physicians are not fully aware that the lesion is fairly common in other areas of the body, especially in the deltoid and gluteal regions following subcutaneous injections. In these areas it may be mistaken for neoplasms. The history of injections or other trauma is helpful in making a differential diagnosis, although surgical excision may be necessary for final diagnosis if the area of fat necrosis does not subside.

Injuries to the lower extremities in our experience are more apt to be associated with traumatic fat necrosis. In such instances the fat necrosis may not resolve for long periods and disability may be accordingly prolonged. This is especially true in obese women with varicose veins in whom trauma to the legs may be followed with indolent ulcers and painful indurated areas of fat necrosis.

SUMMARY

1. One hundred sixty-three cases of fat necrosis collected during a twelve-year period have been reported. No cases associated with pancreatitis have been included.

2. Seventy-four of these patients had the lesion in the breast, sixty-five in or about the peritoneal cavity and twenty-five in the subcutaneous tissue.

3. When the lesion is palpable, it is readily confused with neoplastic growths and must be differentiated from them.

4. The lesion occurs predominantly in females. It has an apparent relationship to some form of trauma in approximately 60 per cent of instances, and to infection in an additional 15 per cent.

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An Experimental Study of the Etiology of Fat Embolism

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Despite the hundreds of articles written on the subject of fat embolism since the seventeenth century, opinions differ as to its clinical importance, methods of diagnosis, etiology and treatment.

We believe that fat embolism is common in persons suffering from injuries and that the effects may be serious. We previously reported a study of 109 consecutive patients admitted to the hospital with injuries.[7] In one-half fat embolism was diagnosed by the finding of fat in the urine; 10 per cent of the severely injured died from the effects of fat embolism. We suggested that the demonstration of free fat in the urine was a practical and reasonably accurate test for fat embolism. Peltier[8] has described the demonstration of fatty emboli in the blood by means of phosphin 3R.

The treatment of fat embolism has been non-specific and supportive. To develop better treatment we must learn more of its etiology. In the studies reported herein we were concerned primarily with the origin of the fat causing fat embolism. We also evaluated the treatment of fat embolism with heparin, theoretically a possible specific agent.

Three possible sources of the fat in fat embolism have been suggested.

Mechanical. The concept that fat liberated at the site of trauma gains access to open veins has been popular. Extraneous and even particulate matter can enter the veins as evidenced by numerous reports of bone marrow embolization.[3,13] However Lehman,[6] from studies of the effect of injecting cottonseed oil into dogs, calculated that all the fat in a man's femur was not enough to kill him.

Humoral. Fatty emboli may come from the physiologic emulsion of fat normally circulating in the blood.[2,6] Normally, chylomicrons and other formed elements of the blood are mutually repellent and stabilized in suspension.[2] The stability of the suspension depends on the fact that each particle has a coating or shell of protein and together the particles form a protein dispersion. Protein dispersions are stabilized by the charge and the hydration of the proteins. The stability of the dispersion might be broken either by neutralization of the charge or by dehydration of the protein. It is possible that stress may release cations, such as calcium and magnesium, or histamine which would neutralize the charge. Stress may also mobilize lipids. This would result, in turn, in increased lipolysis, increased fatty acids and decreased hydration of the protein. In either case embolism or thrombosis might result from agglomeration of the particles.

Trigger. Fatty emboli may come from the physiologic emulsion of fat in the blood in another way. It is possible that the entrance of a small amount of fat into the veins acts as a trigger to break the emulsion causing agglomeration of the particles.

METHODS

The following studies were carried out to evaluate the aforementioned concepts.

1. To determine if the marrow of the femur of a rabbit had sufficient fat to kill the animal, we injected the marrow from the femur of one rabbit into the ear vein of another rabbit of the same size. The marrow was removed from the femur of the donor rabbit and mixed with 5 cc. of saline plus one drop of heparin solution. This was centrifuged to remove blood, bone spicules and fibrous tissue. The supernatant fat was then aspirated into a syringe for injection. Ten rabbits were used in this study. They weighed about 3.5 kg. and their femurs con-

tained about 2 ml. of fat. This was injected during a period of about five minutes.

To compare the lethal effect of corn oil with that of marrow fat, this experiment was repeated, administering 2 ml. of corn oil intravenously to ten rabbits.

2. We would expect a humoral mechanism for fat embolism to take a longer time than a mechanical mechanism. To determine how soon fat embolism could occur after injury, six rabbits were killed immediately after having both femurs fractured by multiple blows from a hammer. They were killed by a crushing blow to the head, after which they were examined for evidence of fat embolism.

3. To determine if the adrenals were essential for the development or occurrence of fat embolism, adrenalectomy was performed immediately before the femurs were fractured in another group consisting of six rabbits.

In a related study massive doses of ACTH* (120 to 600 mg. per kg.) were given to four rabbits and cortisone† (120 to 600 units per kg.) was given to four rabbits to determine if either drug might cause fat embolism. These substances were given in four divided doses within twelve hours, and the rabbits were sacrificed with pentothal® twenty-four hours after the start of the experiment. They were then examined for evidence of fat embolism.

4. It has been suggested that shock may initiate, aggravate[4] or simulate[14] fat embolism. To determine if hemorrhagic shock aggravated fat embolism in rabbits, the animals were bled of half their blood volume, after which both femurs were fractured.

5. To determine if a significant amount of fat was mobilized from the marrow after fracture, stained fat was injected into the marrow cavity of tibias of ten rabbits. The tibias were fractured the following day. Both corn oil with a low melting point and hydrogenated cottonseed oil (Crisco®) were used. Hydrogenated cottonseed oil was used because it more closely resembles human marrow fat in melting point and viscosity.

6. Heparin has the ability to clear the turbidity caused by the fatty particles in lipemic serum.[1,2] Theoretically, it seemed that heparin might well be effective in the treatment of fat embolism despite the work of Becker, Bowman and Grossman[1] who reported it increased the mortality from experimental fat embolism in rats. They administered 10 mg. per kg. of heparin intravenously before introducing 1 ml. per kg. of corn oil into the tail vein of rats.

We thought that heparin might show the irregular series effect and it might be beneficial in smaller doses than those used by Becker, Bowman and Grossman. From the clinical standpoint we thought it more practical to give the heparin after, rather than before, producing fat embolism. We gave heparin intravenously to twenty-four rats in varying doses of 2 to 10 mg. per kg. immediately after injecting 1 ml. per kg. of corn oil into the tail vein.

* Supplied by Parke, Davis & Co., Detroit, Michigan.
† Supplied by Merck & Co., Inc., Rahway, New Jersey.

TABLE I
SYSTEM FOR GRADING OF FAT EMBOLISM BY MICROSCOPIC EXAMINATION OF LUNG

0 = No emboli in entire section (about 1 sq. cm.)
1 = 1 to 4 emboli in entire section
2 = 5 to 10 emboli in entire section
3 = Emboli in each low power field
4 = Many emboli in each low power field

In order to compare the results from these various experiments, a system for grading the severity or degree of fat embolism by microscopic examination of the lungs was devised. (Table 1.) The lungs were selected for study because they are the most frequently involved of all the organs.

RESULTS

1. Injection of the fat from the marrow of the femur of one rabbit into the ear vein of another rabbit of equal size resulted in death in nine of ten animals. (Table II.) Injection of an equal amount of corn oil resulted in no deaths.

TABLE II
COMPARISON OF EFFECTS OF FAT FROM ONE FEMUR WITH ADMINISTRATION OF AN EQUAL AMOUNT OF CORN OIL*

Procedure	No. of Rabbits	No. of Deaths
Marrow fat from femur injected intravenously (2.0 ml.)	10	9
Corn oil injected intravenously (2.0 ml.)	10	0

* This test was performed in rabbits weighing about 3.5 kg.

2. Twenty-four hours after fracture of both femurs an average grade 3 fat embolism was found. (Table III.) This represents severe fat embolism. In animals killed immediately after fracture of both femurs approximately the same amount of fat embolism was found.

TABLE III
TEST OF THE HUMORAL THEORY*

Procedure	Average Grade of Fat Embolism
Fracture of both femurs, sacrificed 24 hr.	3
Fracture of both femurs, head crushed immediately	2+
Femoral fractures after bilateral adrenalectomy	2

* This theory was tested by determining how soon fat embolism occurred and if the adrenals were necessary for the development of fat embolism.

TABLE IV
RESULTS OF AN ATTEMPT TO CAUSE FAT EMBOLISM BY ADMINISTRATION OF ACTH AND CORTISONE TO SIMULATE STRESS

Procedure	Average Grade of Fat Embolism
Cortisone (120–600 mg./kg. in 12 hr.)	1+*
ACTH (120–600 units/kg. in 12 hr.)	1−
Control (normal saline, 15 cc. in 12 hr.)	2

* In one instance there was microscopic evidence of fat cell embolization. The cell probably entered a vein after being dislodged by the needle.

3. Removal of the adrenals did not prevent the development of fat embolism, as shown by the finding of grade 2 fat embolism after fracture of the femurs. (Table III).

Table IV shows the results from the administration of cortisone and corticotropin. The animals in this study showed some fat embolism but not to the degree of those in which the femurs were fractured. The controls had a higher grade of fat embolism than did the hormone-treated animals. We found a fat cell embolus in one animal treated with cortisone. Only mechanical injury of fatty tissue with liberation of the cell and its entrance into a vein could explain the presence of this cell in an arteriole of the lung. The cell must have been dislodged by the needle used to inject the cortisone since the injection was the only injury.

FIG. 1. Lungs of a rabbit forty-eight hours after injection of green stained fat into the tibial bone marrow cavity and twenty-four hours after fracture of the tibia. The fat may be seen as fairly extensive gray mottled areas.

4. Less fat embolism was found in rabbits in which fat embolism was induced by femoral fractures while the animals were in shock. (Table V.)

TABLE V
EFFECT OF HEMORRHAGIC SHOCK ON EXPERIMENTAL FAT EMBOLISM

Procedure	Average Grade of Fat Embolism
Femoral fractures (control)	3
Femoral fractures after hemorrhagic shock	1

5. Large amounts of stained fat appeared in the lungs after fracture of femurs into which stained fat had been introduced. (Fig. 1.) Stained globules of fat were easily seen on microscopic examination of frozen sections. When a fat with a low melting point was used, such as corn oil, more fat embolism occurred than when a fat with a higher melting point,

such as hydrogenated cottonseed oil (Crisco), was used. (Table VI.) Hydrogenated cottonseed oil more closely resembles human bone marrow fat with regard to melting point and viscosity. The controls showed a moderate amount of fat

TABLE VI
COMPARISON OF AMOUNT OF FAT EMBOLISM AFTER INJECTION OF STAINED FATS INTO MARROW CAVITY OF TIBIA

Procedure	Average Microscopic Grading	
	Corn Oil	Crisco
Fracture	4+	3+
No fracture (control)	2+	2

TABLE VII
EFFECT OF HEPARIN ON EXPERIMENTAL FAT EMBOLISM IN RATS*

Procedure	Mortality
Corn oil	2 (8)†
Corn oil + 2 mg. heparin/kg.	2 (8)
Corn oil + 4 mg. heparin/kg.	3 (8)
Corn oil +10 mg. heparin/kg.	4 (8)

* Experimental fat embolism was produced by administering 1 ml./kg. of corn oil intravenously.

† Number in parentheses is the amount of persons in the test.

embolism, demonstrating the vascularity of the marrow cavities.

6. The results of treating experimental fat embolism with heparin are shown in Table VII. Heparin, in the doses used, increased the mortality.

COMMENTS

Lehman's[6] conclusion that there is not enough fat in the femur of a man to kill him has been quoted frequently. If this is true, where does the fat which causes fatal fat embolism come from? We approached the question raised by Lehman directly by injecting the fat from the marrow of a femur of one rabbit into the veins of another. Of ten rabbits in which this was done, nine died. The rabbits weighed about 3.5 kg. and their femurs contained about 2 ml. of fat. The injection of an equal amount of corn oil caused no deaths. The animal fat was more lethal. This is in agreement with previous observations as discussed by Scuderi,[11,12] among others. Marrow fat contains much olein and Scuderi[12] believes that oleic acid derived from the olein by hydrolysis may be the lethal factor. He found that 0.33 cc. per kg. of oleic acid was fatal but 2.2 cc. of olive oil were necessary to produce death. He suggested that the oleic acid may combine with alkalies in the blood to form soaps which are toxic. Warren[13] was unable to demonstrate histochemical evidence of fatty hydrolysis about fatty emboli.

An important consideration is that intact fat cells and other tissue fragments are involved in embolism from bone marrow fat. These do not possess the flexibility of free bone marrow fat or its experimental substitutes, corn oil and hydrogenated cottonseed oil. The tissue fragments or intact cells are more likely to cause a firm occlusion of larger vessels than the more pliable free fat that may be passed through the capillaries of the lung into the systemic circulation.

That there was sufficient fat in the femur of one rabbit to kill another of the same size suggests that there is enough fat in the marrow of the femur of any animal to kill it. Recently Peltier[9] has demonstrated more fat in the femur of an adult than Lehman had estimated there would be. Peltier found 121 gm. of fat in the femur of an eighteen year old and 172 gm. of fat in the femur of a sixty-five year old man, in contrast to the 65 cc. which Lehman estimated. From the data of which the aforementioned is a part Peltier writes, ". . . it would appear that there is sufficient fat in the long bones to account for the phenomenon of fat embolism; the source of the emboli being the local site of injury."

We found fatty emboli within seconds after the fracture of both femurs. This agreed with the studies of Peltier,[8] who found fatty emboli in the blood immediately after injury. Warren[13] found that fat embolism occurred immediately in twenty-six of 100 fatal cases which he studied. It is difficult to believe that a humoral response to stress could have been responsible for these emboli because humoral mechanisms act more slowly.

We found no evidence in these experiments to support the humoral theory of fat embolism. However, fat embolism may occur in more than

one form. Perhaps the pathogenesis need not be mechanical *or* humoral but both may be factors with either one playing a dominant role under differing circumstances. Warren[13] credits Bürger for distinguishing three forms of the pulmonary type of fat embolism: (1) the *peracute*, in which death occurs in a few seconds; (2) the *acute*, in which the symptoms appear immediately after injury and death occurs in hours to days; and (3) the *subacute*, with a free period between injury and the appearance of symptoms. The pathogenesis of the several types may differ. We have observed agglomeration of cells, platelets and chylomicrons after fatty meals in animals, and it is possible that minor fat embolism occurs after fatty meals or stress. This would explain the finding of some fat embolism in all of our animals, including the controls. In fulminating or peracute fat embolism the dramatic lethal aspects may overshadow and divert attention from embolic or thrombotic phenomena.

We found that fat embolism followed femoral fractures even in the absence of the adrenals. It would appear that the function of intact adrenals is not a necessary part of fat embolism. We were unable to produce significant fat embolism by administering massive doses of corticotropin or cortisone in an effort to simulate stress. These results confirmed our previous studies[3] but failed to support the stress or humoral theory for the etiology of fat embolism.

Harman and Ragaz[4] found that hemorrhagic shock increased the mortality from experimental fat embolism. Whitson[14] has suggested that the histologic findings in persons dying from shock are the same as those described for fat embolism. We found that when hemorrhagic shock was produced before fat embolism, it decreased rather than aggravated the fat embolism which followed femoral fractures. We have no explanation for the reduced fat embolism which we found following hemorrhagic shock. Possibly less fat was carried to the lungs because of the impaired circulation. The results suggest that hemorrhagic shock and fat embolism are not directly related.

Introducing stained fat into tibias before fracturing them was an unnatural procedure. However, under these conditions a large amount of fat was transported from the marrow to lodge in the vessels of the lungs. This confirmed the findings of Busch and others, as reviewed by Scuderi.[11] Fat can be carried from the marrow cavity to the lungs by way of the veins. The recent work of Harrison and Gossman[5] is also in accord with these findings. They found that radiopaque suspensions introduced into cancellous bone in the extremities of the cadaver are rapidly removed into the venous system. We found that less fat embolism occurred with hydrogenated cottonseed oil than with corn oil. The cottonseed oil more closely resembles animal fat in melting point and viscosity.

Peltier[10] supports the mechanical theory of fat embolism. He states, "Although in cases of fat embolism there is some debate as to whether all of the embolic fat originates from the point of local trauma, there is no doubt that a considerable portion enters the blood stream at this point." He suggests that the most important manner in which tissue aggregates reach the vascular system to cause "parenchymatous embolism" is "intravasation," the opposite of extravasation. He presents evidence that intravasation can occur in the presence of three factors: (1) a fragmentation of the parenchyma, (2) a pervious vascular bed such as that in the marrow after a fracture, and (3) a shift in the hydraulic equilibrium which is most sensitively balanced in tissues which are confined by bone, a rigid capsule or a contractile muscular coat.

We hoped that heparin might prove to be the specific treatment for fat embolism we were seeking because of its ability to clear lipemic serum. Numerous substances have been used for the treatment of fat embolism.[3] The only treatment which approaches being specific is the intravenous administration of 5 per cent ethyl alcohol in 5 per cent dextrose in water, as suggested by Herrmann in his discussion of our earlier studies.[7] Heparin clears the turbidity of lipemic serum. Basically, this means it causes the disappearance of chylomicrons large enough to scatter light and causes the reduction in size and lipid content of the lipoproteins. Although lipolysis is accelerated with the production of glycerol and fatty acids it is probably of less importance than dispersion in the clearing effect.

We found that heparin, instead of protecting against fat embolism, increased the mortality. Becker, Bowman and Gossman, using 10 mg. of heparin per kg., found fatty emboli in equal amounts in the treated animals and in the control animals with a higher mortality in the

treated animals. We found less mortality with smaller doses than with larger doses, but not less than with no treatment. We have no explanation for this.

SUMMARY AND CONCLUSIONS

1. The fat in the marrow of the femur of one rabbit was sufficient to cause fatal fat embolism when injected intravenously into another rabbit of similar size.

2. Fat embolism occurred seconds after fracture of femurs.

3. Fat embolism occurred after fracture of femurs in the absence of the adrenals. Significant fat embolism did not occur from the administration of corticotropin or cortisone.

4. Less fat embolism was found in rabbits if their femurs were fractured while the animals were in hemorrhagic shock.

5. A significant amount of fat was transported from the marrow cavity of a fractured bone to the lungs. The amount was partially dependent upon the physical properties of the fat, less fat embolism occurring with hydrogenated cottonseed oil than with corn oil.

6. Heparin did not prove to be as specific for fat embolism as was hoped. It increased rather than decreased the mortality from fat embolism.

7. Mechanical fat embolism did occur under the conditions of our experiments.

8. We failed to find support for the humoral or trigger theories of fat embolism in these studies.

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DISCUSSION OF PAPERS BY DRS. HALE AND WEIKSNAR, WATTIKER, MERSHEIMER AND WINFIELD, ROBERTS, REED AND FITTS, AND GLAS, GREKIN, DAVIS AND MUSSELMAN

Oscar Perry Hampton, Jr. (St. Louis, Mo.): Discussion of the problem of tetanus is probably worth while from the aspect of therapy. My remarks will be based, as they were before this group two years ago, on my experience at St. Louis Children's Hospital.

The course of therapy of tetanus is designed to save life. We treat many conditions in which we measure our results not only by the saving of life but also the prevention of permanent disability. In tetanus our results are measured by the saving of life alone.

Death from tetanus is not a result of the disease itself but of the complications, the principal death-producing complication being pneumonia. So with this knowledge, therapy has been directed toward the prevention of death by several measures in addition to the specific therapy of antitoxin. Some of these measures are not in complete agreement with what Drs. Hale and Weiksnar stated in their paper.

The basis of therapy is sedation. At St. Louis Children's Hospital experience has shown that avertin,® the basal anesthetic, given rectally is the best sedative; the margin of safety with avertin exceeds that of sodium pentothal and other sedatives.

It is given in dosage calculated on the basis of body weight, but to an extent which will prevent convulsions. To supplement sedation, there is a cardinal rule that nothing be administered by mouth because if there is nothing in the stomach, the danger of vomiting and aspiration-produced pneumonia will be greatly decreased. This means, of course, that all fluid and food intake is by vein, but during the critical period the caloric intake is a minor problem; it is the fluid and electrolyte balances which count.

Tracheotomy has been almost routine. It was performed in a series of thirteen or fourteen consecutive cases, with no mortality.

Excision of the wound itself, or the wound thought to be the cause, is performed only after adequate sedation so that the manipulation of the patient will not lead to a convulsion. In 20 per cent of the cases the wound of entry could not be found. Of course, that created no problem.

In my experience the dose of antitoxin has been given as 40,000 to 50,000 units intramuscularly, and another 40,000 or 50,000 units intravenously which corresponds to the therapy recommended by Drs. Hale and Weiksnar. Administration of antitoxin has not been repeated. It has been demonstrated that there is enough antitoxin in the blood weeks later to counteract even an overwhelming dose of toxin, so there is no reason seen to repeat it. As a matter of fact, Dr. Cook at St. Louis Children's Hospital has demonstrated that even two or three weeks after the massive dose of antitoxin for therapy, active immunity could not be established by tetanus toxoid.

Roswell K. Brown (Buffalo, N. Y.): I appreciate very much the study of Drs. Hale and Weiksnar on this confusing subject, especially the definite recommendations as to how we should manage our patients. I have three questions.

Most of us have been brought up to believe that the immunity reaction is an antibody, antitoxin reaction with the exotoxin of the tetanus bacillus which is neutralized by the antibodies we administer to the patients with the horse serum or the cow serum. I wonder why mild cases of tetanus do not appear after prophylactic doses of tetanus antitoxin.

They seem conspicuous by their relative infrequency. The authors mentioned the lack of antitoxin in the patient who has had clinical tetanus, so my first question is, could more be said about various types of immune reaction in the body's protection against tetanus, other than the antitoxin?

The second question is a practical one with respect to the treatment of the wounded patient. The authors pointed out quite clearly, I believe, that giving tetanus toxoid along with or soon after the administration of tetanus antitoxin is not wise because it results in little or no anti-immunization. I would like to ask if giving the toxoid a few days before the antitoxin would not be a reasonable program. For example, if the wounded patient were given a dose of toxoid at the time of primary surgical treatment and the dose of antitoxin were delayed for two or three days, would that be a sensible or a helpful program?

The third question is in respect to serum sickness which has such a large morbidity. Can we expect human serum to have an antitoxin against tetanus? A booster dose to any veteran is supposed to give a high titer of antitoxin. The Red Cross blood banks have been taking blood from veterans all along. Is it too much for us to hope that we can have this material as a by-product of the Red Cross blood banks with or without a prebleeding booster dose of tetanus toxoid?

Lazarus A. Orkin (New York, N. Y.): I believe that the paper by Drs. Wattiker, Mersheimer and Winfield concerns a most timely subject, and I heartily agree with them.

It is obvious that although a great many of the perplexing and troublesome problems associated with infection have been solved, many new ones have been created. Among these are the complications which follow the administration of antibiotics. The list of undesirable effects is long and varied. I wish to discuss briefly three that have occurred following the extraordinarily widespread use of these drugs:

1. The selective elimination of the antibiotic-sensitive forms of many common pathogens and the emergence of resistant variants.

2. Acquired resistance accompanied by a marked alteration of the metabolic activities of bacteria. Decreased rate of growth, changed fermentation of carbohydrates and protein derivatives, and loss of pigment formation, among others, are indications of a damaged enzymatic system and represent a physiologic response of the bacterial cell against the damaging drug. Although diverse opinions exist concerning the long-range significance of the emergence of microbial strains resistant to our present antibiotics, the development of such resistance is undesirable since it usually ends the usefulness of treatment with the particular antibiotic.

3. The frequency of infections by organisms which were unknown a few years ago. A remarkable sudden increase in the incidence and pathogenicity of Aerobacter aerogenes was reported from the Urologic Service at Beth Israel Hospital in New York by Wilhelm and myself in 1949. An analysis of consecutive admissions to the Urologic Service in 1940 and 1941 was compared to a similar analysis between 1945 and 1948. Thus, during 1940 to 1941 there were twelve instances of Aerobacter aerogenes in 163 patients (7.4 per cent) as compared to 1945 to 1948 when there were 126 instances in 257 patients (49.2 per cent). Subsequent studies of 100 consecutive admissions revealed that Aerobacter aerogenes were present in 75 per cent of the patients at some time during their hospital stay.

We were also concerned with the pathogenicity of this organism which was formerly believed to be non-pathogenic, for the most part. To date, in our hospital bacteremia caused by Aerobacter aerogenes was proved in thirty-two patients, eight of whom died. The organism was frequently recovered in pure culture from the infected organ, excised surgical specimens or infected wounds. These observa-

tions afford ample proof that Aerobacter aerogenes are now usually pathogenic in the urinary tract, often giving rise to grave and at times fatal infection. This problem has not been confined to any one hospital or city.

We should rapidly become aware that we must discriminate in the use of antibiotics and that we must selectively administer them to patients whose cultures reveal susceptible organisms, so that these valuable chemicals will not be used in needless prophylaxis or in the treatment of trivial concurrent infection. One might add that the spread of resistant strains or the "physiologic crippling" of organisms may be prevented by the use of antibiotics only when there is a specific indication for such therapy, and then only in full therapeutic dosages. The currently prevalent trend to administer them for prophylactic purposes may well lead to further unfortunate circumstances in the future.

Howard Errol Snyder (Winfield, Kan.): In the paper by Drs. Hale and Weiksner the statement was made that individuals having toxoid immunization as long as ten years ago still show adequate response within four to seven days by means of antibody formation.

I think it has been shown that a certain small percentage do not show adequate response; certainly in those cases antitoxin as well as toxoid should be given.

I should like to ask the authors whether or not a double dose of the toxoid at the time of prophylactic antitoxin will yield some antibody response.

The paper by Drs. Wattiker, Mersheimer and Winfield raises a question as to whether or not adequate prophylactic antibody therapy was carried out in the cases mentioned.

One case I recall vividly was that of a college president with acute suppurative appendicitis who left the hospital early because he had to make a commencement address. He returned five to seven days later with evidence of a localizing abdominal abscess in the right lower quadrant. On further antibiotic therapy with the same antibiotics—penicillin and streptomycin—the abscess cleared up.

Concerning the cases of Drs. Wattiker et al., if the organism was not sensitive to the antibiotic, why was the infection delayed? Certainly the fact that it came on many days later would indicate that originally the organism might have been sensitive to the antibiotic. Had the antibiotic been used in sufficient dosage and over a sufficient period of time, the delayed infection might not have occurred.

I think this infection must be distinguished from those which develop at the time of antibiotic therapy from organisms which are not sensitive to the drug and are given "free play" when other organisms are suppressed.

Carlo Scuderi (Chicago, Ill.): A number of years ago I did a considerable amount of experimental work on fat embolism and therefore am in a position to appreciate the excellent research work presented by Drs. Glas et al. on the etiology and possible treatment of fat embolism.

Fat embolism is a very difficult condition to analyze correctly. It has many facets to it, some of which lead one far afield.

It has always been my belief that fat embolism is a mechanical problem. Just where this fat comes from is not clear. Some men believe that it is the breaking down of the normal emulsion in the blood stream, while others believe it is the liberation of the fat from the bone marrow of the broken bone.

Personally, I have always believed that there is not enough fat even in the entire femur of an adult to produce fatal fat embolism in a clinical patient. I did considerable work on this phase of the problem in dogs and showed, at least to my satisfaction, that a dog could well tolerate all the fat which was chemically extracted from his own femur, without fatality.

It is a normal physiologic process to have fat in the blood stream held in very small particles called "chylomicrons." These are especially seen after the ingestion of a fatty meal or in a lactating woman. Centrifuge specimens of either type of case will show a supernatant liquid containing almost pure fat droplets and this might occupy 25 per cent of the entire centrifuge tube. The separation is much like cream on the top of a milk bottle. The fact that these people do not develop any clinical manifestations of fat embolism shows that the small fat droplets, as long as they do not coalesce, are well tolerated by the patient.

Experimental work shows that it is possible, by some form of change in the physiologic properties of the fat emulsion in the blood, to produce a coalescence of these small droplets so that large enough droplets are created to cause temporary or partial obstruction of the small capillaries and produce clinical manifestations of fat embolism. Some people believe it may be an enzyme-splitting product from damage to soft tissues with the liberation of histamine or histamine-like preparations. Others believe that it may be a change in the electrical potentials of these particles. I have had the opportunity of studying the mechanical aspects of intervascular fat in the webs of a frog's hind leg. The small fat droplets are propelled along the capillaries with each heart beat and become elongated, then slowly pass from the arterial to the venous side of the circulation. At no time does actual true stoppage of the circulation occur in an embolus. Many years ago Bissell showed that the viscosity of the blood was changed materially by addition of droplets of oil.

When you produce a retardation of the circulation in the vital centers, such as the brain, for a

period of three to five minutes, irreversible changes occur which may produce death.

The authors of this paper have done an excellent scientific job and I hope they will continue their fine work on this very interesting but puzzling problem.

PETER B. WRIGHT (Orlando, Fla.): The paper by Drs. Wattiker, Mersheimer and Winfield certainly sounded a note that to me is very important.

Not long ago we saw a patient with a note from her doctor to the effect that she had been given 200,000 units of penicillin and ¼ gr. of morphine. She had two open fractures of the femur, a penetrating wound of the chest and a serious head injury.

By simply administering large quantities of penicillin or another antibiotic to our patients, we make it difficult for the bacteriologists to determine sensitivity. Bacteriologists have proved that organisms multiply by mitosis and can quickly develop into variables under the influence of antibiotics. In doing so, one strain of bacteria may be sensitive; the other not sensitive. We may come in contact with a bacterial strain that is so far foreign to the original that we cannot find an antibiotic to which the organism is sensitive. If we will wait for the culture, sensitivity tests will then enable us to use antibiotics correctly.

MICHAEL LIVINGOOD MASON (Chicago, Ill.): I would like to comment on Drs. Wattiker, Mersheimer and Winfield's paper on antibiotics.

I think it is obvious that we do not know everything about this environment of bacteria in which we live and have become accustomed to living.

Antibiotics are now almost universally distributed. They are apparently changing the bacterial environment, and we have to learn how to live with these bacteria again.

I have had no experience with the type of cases reported by these authors. However, I have had three or four patients within the last year or so who have been treated for hand infections in the early stages before a culture could be made. The therapy used consisted of warm packs, elevation and bedrest, and administration of an antibiotic, usually penicillin.

At the time of drainage of these infections, which cleared up very satisfactorily, cultures and sensitivity tests were made. It was surprising to find that these infections, which subsided very well with warm packs, splinting and drainage, were due to organisms not sensitive to the antibiotic used.

ROBERT MAYO TENERY (Waxahachie, Tex.): I would like to ask Dr. Hale two questions. Does he think that the administration of 1,500 units of tetanus antitoxin prophylactically is really of any value? Also, does he believe that erythromycin has any value prophylactically or therapeutically in tetanus?

HARRY W. HALE, JR. (closing): I would like to thank the discussers of our paper. I am sorry Dr. Hampton did not get into the question of immunization because I believe he probably would have disagreed with us again.

I do not think we are too far apart on the points he mentioned. I certainly would not argue about the drug used for sedation. There are many different ones. The important point is adequate sedation.

As far as tracheostomy is concerned, I will go along with the suggestion that it should be almost routine.

As to Dr. Brown's questions, if I interpret them correctly, I believe the first one implies that the subject I have discussed with him is that the circulating antibiotics we measure in the test tube represent only one phase of the immune reaction of the individual. Unfortunately, this is the only one we can conveniently measure. There are other known and unknown mechanisms involved, apparently, which may or may not actually parallel the level of circulating antibodies. Of course we can only measure these latter aspects of the immune process or immunity of the individual more or less roughly and indirectly by clinical responses.

I do not know whether toxoid given one day or several days before antitoxin would be of any greater value than when given simultaneously. It is known that the response to a primary dose of toxoid is a very slow one, and I think perhaps several days would have to elapse although that, of course, is just a guess.

Use of human serum globulin has been recommended by Dr. Stafford, particularly, and I think it would be very valuable. It has been known that tetanus antibodies are concentrated highly in the human globulin of an individual who has been immunized recently or had a recent booster. I believe this should be generally adopted.

I was not aware that lack of response to a booster dose had been found in individuals immunized over ten years ago. That is a very important finding.

The size of the dose of toxoid in relation to its inhibition or interference by passive antibodies given at the same time has been studied, as nearly as I can find, only in limited series and in limited amounts. Apparently doses up to two or three times the ordinary toxoid dose do not seem to influence the inhibition.

The whole question of inhibition of toxoid in its effect by passively introduced antibodies is apparently one of ratio of concentrations. We know that diphtheria toxoid and antitoxin are given at the same time. For many years they have been used and are known to produce effective, active immunity, and the exact relationship of dosages and

amounts in tetanus may be worked out some day so that we can safely perform the same type of immunization in this disease. So far I do not believe we have solved that problem.

Dr. Tenery questioned the value of 1,500 units of antitoxin. I think it is of some value, although limited. I did not mention, and should have, that whatever dosage is used primarily its value will only last somewhere between seven and fourteen days, and in instances of infected wounds or wounds that remain open where the danger of tetanus contamination continues, antitoxin must be repeated to be of any value, preferably every seven to ten days.

There are many who recommend much larger dosages than this, but in the average individual, 1,500 to 3,000 units will give a satisfactory level, and that is interpreted by authorities as being .1 units per milliliter of circulating serum as an adequate protective level for passive antibodies.

I am sorry I cannot answer the question about the value of erythromycin used either therapeutically or prophylactically.

BERNARD J. WATTIKER (closing): We are in agreement with Dr. Wright that repeated cultures and sensitivity studies are often necessary in treating patients with bacterial infection.

In answer to Dr. Snyder's question regarding the sensitivity of various organisms, we cannot answer it specifically inasmuch as we did not take cultures preoperatively on any of these patients, and we do not know whether or not there was inoculation of sensitive organisms or insensitive organisms, or whether these organisms later acquire a resistance under treatment. However, there are many theories postulated as to the occurrence of these infections.

Most people stress the fact that resistance is not acquired, but rather the Staphylococcus aureus that produces it, and that these organisms are resistant from the start. However, there is considerable evidence on the opposite side which states that organisms may acquire resistance during treatment.

We had a case similar to the one which Dr. Snyder stressed. A patient with a ruptured appendix was operated upon and treated with penicillin and streptomycin. Cultures at that time showed sensitivity. However, this patient later returned with a pelvic abscess and was placed on antibiotics again, with no response. Later the patient responded after drainage. At that time it was found that the organisms present were resistant to the antibiotics used.

W. W. GLAS (closing): I would like to thank Dr. Scuderi for his comments on our paper. We recognize him as an established authority on the subject.

He mentioned several things which are of considerable interest to us. The fact that we removed all of the bone marrow fat from the marrow cavities was a distinct reference to Lehman who substituted cottonseed oil for bone marrow fat in dogs and concluded there was insufficient fat to produce fatal fat embolism. If he had used the bone marrow fat, as we did, I am sure he would have experienced results similar to ours. In rabbits the entire amount of femoral bone marrow fat is not required to cause fatal fat embolism. Less than half this amount is lethal.

The fact that some fat is mobilized from the site of trauma cannot be questioned inasmuch as many of you, I am sure, have seen intravascular bone spicules in the lungs on microscopic examination of suspected cases of fat embolism. These bone spicules are naturally filtered out in the lungs. This implies that the bone marrow fat has a similar origin and fate. This also explains why the lungs are more frequently and universally involved than any other organ.

With regard to the concept of destabilization of protein and the loss of electrical charge, we have looked extensively throughout the systems of organs of both human and experimental animals, hoping to find evidences of fat embolism in the kidneys, the brain, the liver or the heart which would equal that found in the lungs. We believed that if there were a destabilization effect it should be found on the arterial side as well as on the venous side and in somewhat similar frequency and volume. We have never found fat embolism on the arterial side to equal that on the venous side of the circulation. The fat is found predominantly in the lungs, which leads us to believe it must be picked up by the venous system.

This takes us back again to the subject of properties of fat, which has been discussed by Dr. Roberts. Fat is different in various types of people; it is different in children and in adults. In addition, there is a difference in host resistance; some people will tolerate the same degree of fat embolism better than others.

Recently there has been additional work by Harrison demonstrating the extreme vascularity of the bone marrow cavities. He obtained excellent venograms of an extremity by injecting a radiopaque dye into the bone marrow cavity of the extremity. This occurred within seconds following the injection of the dye.

In closing, I would like to say we are still interested in the etiology of fat embolism, and will continue our work in this field. We agree with Dr. Scuderi completely; we do not know half of the answers we would like to know.

Herniated Lumbar Intervertebral Disks

AN EIGHT-YEAR SURVEY

CARLO SCUDERI, M.D., *Chicago, Illinois*

THE following series of cases was seen from 1947 to 1954, numbering 113 patients with 115 disk operations. Two patients were reoperated upon for recurrent herniated disks.

Only cases with adequate follow-up are included in this series. Cases treated prior to 1947 were not included, as this was a period of metamorphosis for the author. From 1947 on sufficient experience was gained to evaluate the surgical indications, and since that time I have made very few changes in the surgical procedure or aftercare.

The problem of the herniated disk is still unsettled. Only a few of the multiple unsettled phases of this problem are: indications and contraindications for surgery, fusion versus non-fusion, and laminectomy versus interlaminar exposure without bone removal.

Only by frequent discussions and presentations of personal experiences of various orthopedic and neurologic surgeons will this problem ultimately settle into its proper perspective.

The author does not intend to go into the clinical characteristics of this disease, as they are well known; however, the objective findings in this group of cases will be presented because the ultimate figures were surprising to the author. The percentage of reflex and sensory changes in this series was much lower than anticipated, whereas the Lasègue and Kernig tests were much higher. (Table 1.)

TABLE 1
SIGNS INDICATING HERNIATED DISK

	Present		Absent	
	No. of Cases	Per cent	No. of Cases	Per cent
Reflex changes (Achilles tendon, patellar)	68	59.1	47	40.9
Sensory changes	72	62.6	43	37.4
Atrophy	106	92.1	9	7.8
Lasègue's sign	112	97.4	3	2.6
Kernig's sign	111	96.5	4	3.5

There is a great deal of difference of opinion between some men of equal medical standing in the same community as to the diagnostic value of myelography. In all cases in which herniated disk is suspected I am strongly in favor of myelography for the following reasons: (1) The diagnostic accuracy in this series has been 90 to 91 per cent in the cases seen between 1951 and 1954; in medicine any test with this degree of accuracy should certainly be utilized. (2) It localizes the level of the lesion, thereby saving operative time and an extra risk to the patient. (3) If there are multiple herniated disks, the surgeon is forewarned and fewer errors are made. (4) The presence of cord tumors in the diagnostic picture should be kept in mind and can be readily diagnosed in most instances by myelography; if no other indications existed, this one alone would be priceless in many cases.

That cord tumors can be differentiated from herniated disks by accurate history and clinical examination will not be disputed. The exact location of the disk or any multiplicity cannot be accurately diagnosed by the most careful diagnostician, and those who claim this virtue are simply playing mathematical percentages.

Myelographic accuracy in these cases have been divided into two groups: (1) 1947 to 1951

TABLE II
RESULTS OF ROENTGENOGRAPHIC AND MYELOGRAPHIC EXAMINATIONS FROM 1947 TO 1951*

Results	Study of Disk	No. of Cases	Per cent
Positive	Positive	37	72.5
Positive	Negative	9	17.6
Negative	Positive	3	5.9
Negative	Negative	2	3.9

* No. of total operative cases 51. Accuracy of 76.5%.

(Table II), which was the learning period of the author and the radiologist who worked with him, and (2) 1951 to 1954. (Table III).

In this series the L4–L5 interspace had slightly more herniations than the L5–S1 despite the fact that the greatest curvature and

TABLE III
RESULTS OF MYELOGRAMS FROM 1951 TO 1954*

Results	Study of Disk	No. of Cases	Per cent
Positive	Positive	55	87.3
Positive	Negative	3	4.7
Negative	Positive	3	4.7
Inconclusive myelograms	.....	2	3.2

* Total number of operative cases, 62. Accuracy of 90.4%.

TABLE IV
LEVEL OF HERNIATED DISK

Level of Disk	No. of Cases	Per cent
L5-S1	52	47.7
L4–L5	55	50.5
L3–L4	2	1.8

TABLE V
LOCATION OF HERNIATED DISK

Location	No. of Cases	Per cent
Left side	56	51.4
Right side	46	42.2
Central	7	6.4

stresses and strains of the lower back appear to be at the L5–S1 level. (Table IV.) Table V lists the location of the herniated disk.

Formerly the author followed the teaching that all disks should be fused, but as time went on it was realized that spinal fusion is an additional major operation. It prolongs the operating time, adds more postoperative shock, lengthens hospital stay, prolongs convalescence and, if that were not enough, means an additional operation which in itself carries a 15 to 35 per cent incidence of failure.

The average patient with uncomplicated disk without fusion is able to do light work in four to six weeks and heavy work in eight to ten weeks. The average patient with fusion is incapacitated a minimum of sixteen to eighteen weeks from the lightest work and twenty-four to thirty weeks from heavy work.

TABLE VI
TYPE OF OPERATION

Results	Disk Alone	Fusion, No Disks	No Disk, No Fusion	Disk and Fusion
Excellent	52	1	1	4
Good	25	2	1	4
Fair	6	2	2	4
Poor	2	0	4	3
Totals	85	5	8	15

At the present time no disk case is routinely fused, and only those cases which fall into one or more of the following categories are considered for fusion: (1) hemilaminectomy or total laminectomy performed in the removal of the disk; (2) spondylolisthesis; (3) spina bifida; (4) sacralization of a transverse process, with evidence of a false joint; (5) marked bony osteophytes in the area; (6) marked marginal sclerosis of the contact bony surfaces just above and below the area; (7) a loose, unstable fifth lumbar vertebra observed at operation and diagnosed by its excessive motion on instrument pressure.

Although the cases in Table VI cannot be evaluated one against the other because of many dissimilar factors, they are presented so the reader may study them and draw his own conclusions.

Many men believe that compensation cases do not do as well as non-compensation cases because of the monetary aspects involved. I have not had this experience. The ultimate outcome of compensation and non-compensation cases for good and excellent end results was almost identical. The figures are presented in Tables VII and VIII.

The key to success in disk surgery can be summed up as follows: accuracy of diagnosis. If the diagnosis is correct and the pathologic condition removed, the outcome is excellent.

If the diagnosis is in error, no disk is found, one is overlooked at a different level, the pa-

tient has a cord tumor or is psychotic, then of course the results are poor.

Overenthusiasm for the surgical cure of backache with or without radiation to the leg will lead to useless, harmful surgery and cause the surgical treatment of herniated disk to fall unjustly into disrepute.

TABLE VII
COMPENSATION CASES*

Results	No. of Cases	Per cent	
Excellent	27	44.3	82.0
Good	23	37.7	
Fair	8	13.1	
Poor	3	4.9	

* Male patients, 58; female patients, 3.

TABLE VIII
NON-COMPENSATION CASES*

Results	No. of Cases	Per cent	
Excellent	31	59.6	80.8
Good	11	21.2	
Fair	5	9.6	
Poor	5	9.6	

* Male patients, 35; female patients, 17.

CONCLUSIONS

1. A careful review of 115 operations in 113 patients with herniated intervertebral disks is presented. Follow-up was from six months to seven years.

2. The diagnostic accuracy of myelography as to the level, side and surgical confirmation is discussed.

3. The indications for fusion and non-fusion are presented, with twenty cases or 17.7 per cent of associated fusions.

4. The end results classified as excellent to good were as follows: compensation cases 82 per cent, and non-compensation cases 80.8 per cent.

DISCUSSION

HOMER HARTMAN STRYKER (Kalamazoo, Mich.): We follow about the same criterion as to indications for fusion that Dr. Scuderi does. I just wanted to add something which we found to be a big help in the last two or three years in doubtful cases. After we expose the interspace, sometimes we find it looks perfectly level and think there is no degenerated disk.

Take a 5 cc. syringe with normal saline, a long No. 23 needle and insert it down into the middle of the interspace and then try to inject the saline. If the disk is perfectly normal, it will take only from 1 to 2 cc. and it will be under pressure and push the plunger back. If it is a degenerated disk, the saline keeps flowing and often it will cause the annulus to bulge up into the canal. After injecting saline and opening the annulus, the disk squeezes out of the annulus like grease out of a grease gun.

This method has been of great aid to us. Since we have used it, we probably take fewer myelograms and diskograms. We can determine whether or not there is actually a degenerated disk and not run the risk of destroying a disk which may have been normal from the start.

ROBERT H. KENNEDY (New York, N. Y.): The Chair would like to ask Dr. Scuderi a question. He said he is an enthusiast about myelograms. Does he believe in taking a myelogram as a diagnostic aid on a patient upon whom he has not decided definitely to operate in the immediate future?

CARLO SCUDERI (closing): Dr. Kennedy has asked a very pertinent question. We always use the myelogram for diagnostic purposes; and whenever there is doubt and we think a patient might have a herniated disk, we take a myelogram and then decide whether or not surgery should be done.

To reiterate, first myelography gives you the level of the pathologic condition; second, whether there are multiple herniated disks; and third, whether there is a cord tumor. If there is, you had best refer it to a neurosurgeon.

The Open Fracture

CIRCUMSTANCES AFFECTING TREATMENT

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Open fractures differ so greatly in the circumstances of the injury that they cannot be considered in a single category regarding treatment.

Army directives in World War II ordered extensive wound excision, loose packing with gauze and suture of the wound only after five days' observation.

Naval medical literature, on the other hand, included a strong opinion by Ferguson et al.[1] that the use of sulfa drugs made radical débridement unnecessary, and that in some instances débridement was actually destructive.

A surgical resident might understandably be confused by these completely divergent views as to treatment, and this paper is intended as a brief summary of the reasons for this divergence.

Let me use my imagination to describe a sailor on a battleship. He is a superb athlete in perfect condition, nourished by ample food and plenty of water and hot coffee, and is required to scrub himself and his clothes and the ship's deck every day. He is in practically sterile surroundings. He is injured by the red-hot shell fragment noted by Dr. Ferguson, and he gets prompt surgical attention.

In contrast, I use my imagination again to describe a soldier in the jungle. He is provided only with D-rations and very little water; he is exhausted by a month in the fighting with no relief, his sleep prevented by an enemy with a bowie knife; he has had no bath for weeks and is smeared with jungle mud as camouflage. The shell fragment that strikes this exhausted and dehydrated individual has ploughed up the jungle mud, well impregnated with feces, and there may, unfortunately, be some delay in getting him to definitive treatment.

Surely the open fractures in these two men bear no resemblance to each other. It is easy to see why the soldier must have the most painstaking wound excision and why immediate suture is dangerous. It is easy to see, as well, that the sailor would run almost no risk of fatal infection and why extensive wound excision would not be mandatory.

To turn to civilian injuries, in ten years in the soft coal mines I saw no case of gas infection, and any infection at all was most unusual. The miners were covered with coal dust but they had to take a hot bath every night in order to be allowed in the house when they went home; the coal and rock had been there uncontaminated since the Jurassic age. I admit there were some rats and mules. Extensive wound excision was never needed although, of course, some trimming was done.

In automobile injuries wound excision is certainly needed, but primary loose suture is ordinarily safe. In nine years at the Veterans Administration Hospital at Miami (Coral Gables) we have seen gas infection only in injuries in the swamps—a total of, I think, three cases.

Railroad and other crushing injuries commonly require amputation; airplane crash injuries are so severe that death is a common result.

In pistol-ball fractures with simple perforating wounds, no wound excision is needed as a rule, and the bone ordinarily heals as a simple fracture.

Let me make reference in passing to the form of treatment that was advocated some years ago by Trueta, a method that ended in few good results and many bad ones. Trueta adapted the Orr method of packing in chronic osteomyelitis to the treatment of fresh open fractures. The trouble was that the two situations are simply not comparable.

In the patient with chronic osteomyelitis the saucerized bone is backed by firm scar, so that suppuration can make no exit except around the vaseline gauze packing. In a fresh open fracture suppuration, walled in by the firm

plug of gauze, easily burrows along loose muscle and fascial planes, with wide spreading of the infection. Sepsis, non-union and amputation were too often the inevitable result.

Of course, the general treatment of the open fracture has been improved in recent years by blood transfusion, a knowledge of the danger of crush injuries, free incision of constricting deep fascia and, I am quite sure, also by the antibiotics.

I believe I should mention here a strong personal opinion that patients with trauma to the extremities should be kept strictly in bed for some days; this method is in contrast to the early ambulation that is so beneficial in postoperative abdominal cases. There is no real paradox here, for in abdominal surgery it is the general condition that must be considered; in trauma to the extremities there is imperative need of keeping the injured part at complete rest for the time being.

SUMMARY

I have intended in this paper to make the point that in training surgical residents it is necessary to call attention to the wide differences between open fractures that at first sight might appear similar; and to the fact that there is no single method of treating these fractures that applies in all circumstances.

Whether or not to employ débridement and how extensively, and whether or not to adopt loose primary closure are questions which can be answered only by thoughtful consideration of the details of the manner of injury and the condition of the injured patient himself.

REFERENCE

1. Ferguson, L. K., Brown, R. B., Nicholson, J. T. and Stedman, H. E. Observations on treatment of battle wounds aboard hospital ship. *U. S. Nav. M. Bull.*, 41: 299–305, 1943.

Massive Sliding Inlay Bone Graft for Correction of Ununited Fractures of Long Bones

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Various types of bone grafting procedures have been utilized in an attempt to correct ununited fractures. Of these, eight main types have been popularly used and described. In 1947 one of the authors (J. H. W.) reported a new technical approach for tibial grafts.[1] In that paper he presented a ninth type of graft, namely, the massive sliding inlay graft which he has been using since 1922. The only changes which have been made since that time have been certain additions and modifications of his original technic. One of these is the dovetailing of the ends of the graft to permit better mortising and impacting of the graft, thereby necessitating less internal fixation. Another change is the shifting of the tibial graft site from the flat anterior to the lateral surface, and a third is a gradual increase in the size of the graft. The other types of grafts referred to previously as those commonly used and accepted are: (1) single onlay cortical graft, (2) double onlay cortical graft, (3) inlay graft, (4) peg graft, (5) intramedullary graft, (6) osteoperiosteal graft, (7) multiple chip graft and (8) whole bone transplant graft.

The purpose of this paper is to bring out the principles, advantages and end results, and show the condition of these patients after long term follow-ups.

TECHNIC

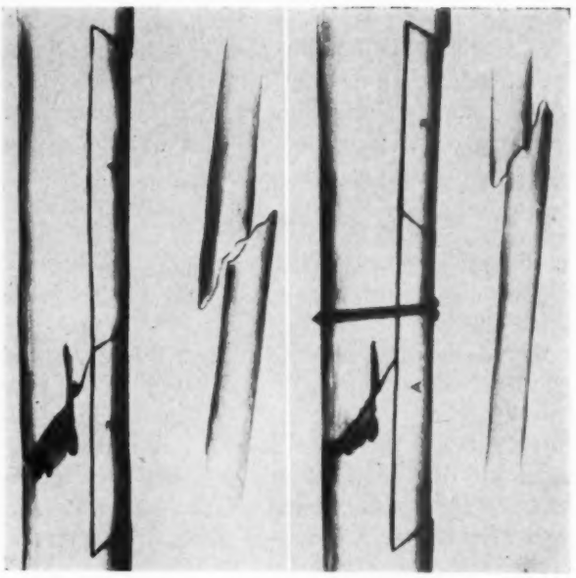
The technic of the procedure will not be gone into in detail but the chief steps of the procedure will be explained. So far as the incision is concerned, it should be large enough to permit good visualization of the bone above and below the site of the ununited fracture. We prefer to use an anterolateral incision for the tibia, femur and humerus, and a dorsolateral and dorsomedial incision for the radius and ulna. In all of these incisions we attempt to separate muscle bundles whenever possible and cut through muscles only when it is absolutely necessary. The periosteum is then incised and the bone is exposed subperiosteally.

When a sinus tract exists or when there is a small ulcerated area near the site of incision, it is our policy to excise this completely at the time that the incision is made. Each incision is made with the thought in mind of getting the best visualization of bone with the least amount of trauma to soft tissue.

After the bone has been exposed, the site of the ununited fracture is completely cleaned out with removal of all fibrous and bony tissue between the bone ends as well as in the surrounding soft tissues. Every attempt is made to get a covering of soft tissue for closure of the wound which is free of calcific deposits or dense fibrous tissue. The bone ends are then freed and the rounded-off, scarred and closed-off portions of the bone involving the ununited fracture are freshened by completely removing the aforementioned tissues. The marrow cavity is then opened into above and below the site of the ununited fracture. This can be done with a knife, curet or bone drill depending upon the density of the tissue. It is only after this step has been carried out that the bone fragments are aligned and the bone graft is outlined. The grafts are then cut *in situ* with the electric saw. (Figure 1A.) Next the grafts are removed, reversed and placed in such a position that the larger graft fits into the mortise, traverses the fracture line and extends far enough above this area to insure good bridging and fixation. The smaller graft is then used to fill in the remaining gap in the bone. (Figure 1B.)

So far as internal fixation is concerned, one must be guided by conditions which are present at the time of the operation. If there is no muscle pull and if the graft impacts easily at its mortised end, a single transfixation screw is sufficient. When the muscle pull is such that a

single screw does not satisfactorily immobilize the graft, a second screw is inserted. If tension is such that too much strain is placed on the mortised end of the graft, a transfixation screw can be used on the opposite side of the fracture line to aid in the immobilization of that end of the graft. In instances in which marked muscle pull is present or in which the extremity has been fixed in an angulated position due to malunion and malposition over a long period of time, it is sometimes necessary to use a bone plate to maintain alignment prior to cutting the grafts. In some instances in which the smaller graft does not readily maintain its position without internal fixation, a single screw can be used. In all instances we have used 18-8 SMO stainless steel Sherman screws and plates for internal fixation.



1A 1B

FIG. 1. A, schematic drawing showing the grafts after they have been outlined and cut *in situ*. Note the dovetail cutting at the proximal and distal ends. B, schematic drawing showing the grafts in position after they have been reversed. The major graft traverses the site of the ununited fracture and is immobilized above this area with a single transfixation screw. The lower end is held by the dovetailing procedure as is the smaller graft.

ADVANTAGES

In our opinion this procedure offers several important advantages over the others commonly used. The foremost is that we have no need for a bone bank. In all of these cases at the time of the first operation we have been able to obtain sufficient bone from the involved bone itself to carry out the bone grafting procedure. By so doing we are using not only autogenous bone but also the involved bone proper. In this manner we are supplying, as a graft, bone which is of the same texture and composition, and which is from the same environment as the bone to be grafted. We believe that this is better than placing a rib in a tibia, a fibula in a tibia, or pieces of bone from the ilium in a tibia or in any other long bone. It undoubtedly is better than someone else's bone which has been frozen for a long period of time.

We also believe that with this procedure we are able to obtain and place across the site of the ununited fracture larger bone grafts which can serve as a matrix for osteogenesis. As the bone in the graft is the same bone involved in the fracture, it is accustomed to its particular environment and is naturally making every attempt to heal. In this manner the healing reaction is set up over an extensive area which then sends blood supply not only into the fracture site but also into the bone graft. With a large matrix to work on, the major fragments of the bone are able to blend the graft into them and form a solid bony mass. This is the type of healing which we have observed.

We have always believed that the medullary blood supply has much to do with whether or not a fracture heals. We have yet to operate on a patient with an ununited fracture in which the fracture ends of the bone were not completely sealed off with either dense fibrous or bony tissue. On the other hand, we have never observed this change on x-ray films in those cases in which the fracture has gone on to healing. For this reason we believe that the procedure has been partly successful because we have always stressed the importance of not only opening up the medullary canal at the site of the ununited fracture but also making every attempt to create and maintain an open intramedullary canal. It will be noted that all of the cases to be described had open intramedullary canals. This is something which is not seen with onlay multiple chip or intramedullary peg-type grafts.

Another advantage is the shape and contour of the bone which are obtained as an end result. There is no marked fullness at the involved site, for the bone heals without bulbous or excess callous formation. The graft blends into the major bone fragments and results in an

almost completely normal-looking bone. No doubt all of you have seen simple uncomplicated fractures of the long bone which have healed without difficulty and which show more fullness at the fracture site than do most of the cases presented in this paper.

BASIC PRINCIPLES

There are several basic principles which must be kept in mind if one is to get good results with this procedure.

Osteoporosis is present in quite a few of these patients when they are first seen. When this condition is present, it is foolish to attempt surgery. Osteoporosis is due to a marked disuse atrophy of the part and is associated with marked demineralization of the bone. Before considering operation it is necessary that the patient use the part actively and that paravertebral blocks and physiotherapy treatments be given so as to remineralize the bone. It is good to wait two or three months if necessary to obtain these changes in the bone before attempting surgery; the results will pay in the end.

Infection in compound fractures is another factor which must be considered. Many of these patients have draining sinuses of long duration quite often associated with sequestrum formation. When drainage is present in these cases it is always necessary to clean out the sequestrum and to obtain complete healing of the sinus tract before surgery is undertaken. In a few instances in which a sinus tract is present without much drainage, one can occasionally successfully excise the tract at the time of the bone grafting procedure. This, however, is risky and should rarely be done.

Of technical importance is the step in the procedure whereby the site of the ununited fracture is completely freed of fibrous and bony tissue and the intramedullary canal is opened into above and below the involved site. We realize that this is a repetition of statements made previously, but it is so important in our minds that we wish to stress it as one of the basic fundamental principles to be observed if success is to be expected.

ANALYSIS OF CASES

From January 1, 1938, to December 31, 1954, 196 bone grafting operations involving 207 bones were carried out on a total of 182 patients.

An analysis of these procedures shows that the bones operated upon were as follows:

Tibia alone	111
Tibia and fibula	1
Fibula only	4
Femur	30
Humerus	11
Radius alone	15
Ulna alone	14
Radius and ulna	10

Of the 182 patients upon whom the aforementioned 196 bone grafting procedures were carried out, 169 resulted in primary bony union following their operations. Complications developed in some patients which prolonged their postoperative period, but healing eventually took place without further surgery. Of the remaining thirteen, eleven required a second bone grafting operation and two others required two other bone grafting procedures. All, however, eventually went on to complete healing. Of the thirteen patients who required more than one operation an analysis shows the following:

Radius and ulna requiring second graft on ulna	3
Femur requiring second graft	1
Tibia requiring second graft	4
Femur requiring two further grafts	1
Radius requiring second graft	1
Ulna requiring second graft	1
Humerus requiring second graft	1
Humerus requiring two further grafts	1

CASE REPORTS

The following cases have been selected from those patients who have been operated upon since the co-author's association with Dr. Wagner in 1942. From time to time interesting cases were noted and follow-up studies were continued. These cases have been selected to show that the procedure can be carried out on patients of all ages, that it can be carried out bilaterally and that most complications which arise can be handled conservatively. In addition, they have been carefully chosen to show the various types of internal fixation which can and have been used. We have made an attempt to choose cases which have the longest period of follow-up.

CASE 1. B. B., a fifty-six year old man, sustained a compound fracture of the tibia and fibula in April, 1943. He was treated elsewhere first by closed and then by open method, with resultant ununited fracture. Grafting was done on February 28, 1944, resulting in primary solid bony healing.

This case is presented to bring out two points: (1) When first seen by us on December 29, 1943, the patient had marked osteoporosis (Fig. 2A) in addition to his ununited fracture. He was made to walk without any external fixation and was given physiotherapy treatments and a series of eight lumbar parasympathetic blocks. It was only after a check film showed that the bone had become remineralized that operation was performed. (2) In this case a single screw was used for fixation of the graft with the lower end being well held by the dovetail technic. The smaller graft fit snugly into the donor site above and required no internal fixation. (Figs. 2A to D.)

Case II. F. P., a fifty-three year old man, sustained a compound fracture of the right tibia and fibula in 1941. He was first treated by closed reduction, then by open reduction with transfixation screw plus Sherman plate fixation. An ununited fracture developed and was grafted in December, 1943. This case is presented to illustrate fixation with two Sherman screws to hold the major graft. As in fractures, we believe in firm apposition and coaptation of the bone fragments. Since the dovetailing procedure *per se* did not give sufficient coaptation, the second screw below the site of the ununited fracture was inserted. The minor fragment was well coaptated without internal fixation. (Figs. 3A to D.)

Case III. H. T., a twelve year old boy, sustained a simple fracture of the left tibia and fibula on June 8, 1943. Treatment consisted of open reduction with plate fixation on June 11, 1943; the injury failed to heal postoperatively. In addition to absorption of bone at the fracture site, the patient also fractured the plate. This case is presented to illustrate the necessity of the transfixation screw in the minor fragment since good coaptation could not be maintained otherwise. In addition, the major fragment was well anchored at the lower end by the dovetail technic, but two screws were necessary to transfix the upper end because of the excessive pull secondary to the bowing which was present. (Figs. 4A to D.)

Case IV. K. B., a forty year old white man, sustained a compound fracture of his left tibia and fibula in November, 1941. He was treated in three other hospitals by closed as well as open methods. At the time of our first examination he had, in addition to his ununited and malunited fractures, a draining sinus over the anterior surface of the left lower leg. This had been draining for more than two years. The patient was first seen by us in July, 1943, and was not operated upon until August 23rd. This case is presented not only to show the importance of preliminary treatment but also to demonstrate plate fixation of the major bone fragments prior to grafting. (Figs. 5A to D.)

Case V. F. M., a seventy-two year old woman, sustained a simple fracture of the upper end of the left tibia and fibula in January, 1951. The leg was placed in a cast, and in May the attending physician recommended open reduction. Following this she was transferred to one of us and a bone graft was carried out instead. This case is presented to show the necessity of fixation of the graft at both ends to overcome muscle pull, and to show that healing takes place quite rapidly even in the older age group. (Figs. 6A to D.)

All of these cases were uncomplicated and went on to primary healing. The following case is one in which the major graft was fractured while being removed from the donor site. The illustrations show how this complication was handled.

Case VI. W. B., a twenty-three year old man, sustained a simple fracture of the upper third of the left radius one year prior to being seen by us. He was treated by closed reduction but the fracture did not unite. (See legend, Figure 7.) This case is included to bring out the fact that even though some technical complication may develop, the procedure can still be carried on satisfactorily. In addition, it also shows how multiple grafts can and do blend into the major fragments of the bone as well as heal to each other. (Figs. 7A to D.)

Case VII. R. L., a seventeen year old white girl, sustained multiple fractures in an automobile accident in January, 1949. Ununited fractures developed in both femoral shafts and were grafted elsewhere. The right graft healed, but the left was unsuccessful and the patient was sent to us. This case is presented to show two complications which may arise and the methods by which they were treated. The first was a fracture of the shaft of the femur proper at the donor graft site and the second was a fracture through the major graft itself at the site of the ununited fracture. (Figs. 8A to H.)

Case VIII. W. S. W., a twenty-one year old woman, sustained a simple fracture of the left femur in January, 1941. She was treated by closed and then by open reduction which resulted in an ununited fracture. She was first seen by us early in 1942 and grafting was done in March, 1942. This case is presented to show a graft formed without the dovetail technic. In this operation the grafts were cut straight across, and more internal fixation was necessary than is used in cases in which dovetailing is carried out. Here, too, the patient had fractured the plate and had marked angulation at the site of the ununited fracture. (Figs. 9A to D.)

COMMENTS

From the foregoing observations, we believe we have made certain points about this procedure which substantiate our feeling that this

2A 2B 2C 2D

FIG. 2. Case 1. A, film taken December 29, 1943, three months before operation. In addition to the ununited fracture of the tibia, there is marked osteoporosis with demineralization and so-called disuse atrophy of the bone. Note also the absence of any callus at the fracture site and the obliteration of the marrow cavity at the area of non-union. The patient was made to bear weight without external fixation and was given physiotherapy and paravertebral blocks prior to his operation on February 28, 1944.

B, film taken February 29, 1944, shows the large graft immobilized with a single Sherman screw. Note the alignment of the bone as well as of the bone grafts. At this time the osteoporosis is much decreased and the operation was carried out on fairly healthy bone.

C, film taken May 6, 1944, nine weeks following the operation. The grafts are becoming incorporated into the tibia and are now healing to each other as well as blending in with the major tibial fragments. Some osteoporosis is still present.

D, film taken April 14, 1954, ten years and two months following the operation. There is now complete bony union and one has difficulty in noting that this bone was grafted. One cannot tell the graft site, and the end result is comparable to that of a well healed fracture. There is no excess callus, no gross deformity and the medullary canal is open throughout.

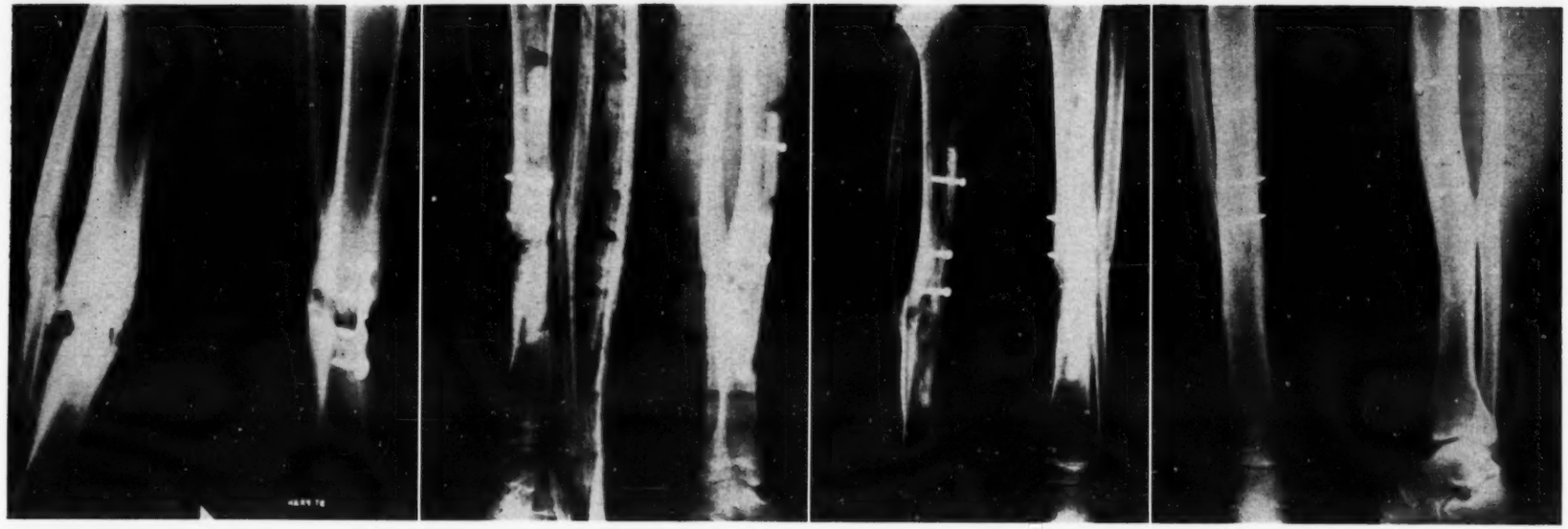
3A 3B 3C 3D

FIG. 3. Case 11. A, film taken December 28, 1943, shows the ununited fracture of the tibia with absorption around the transfixation screw and with loss of bony tissue at the fracture site. Alignment is satisfactory but there is no evidence of healing.

B, film dated May 18, 1944, shows almost complete healing of the grafts to each of the major tibial fragments. There is still a space between the grafts and the fracture is still partially ununited.

C, film dated June 13, 1946, shows complete healing two and one-half years following the bone grafting procedure. Here the fracture is completely healed and it is difficult to differentiate clearly the area where the grafting procedure was performed.

D, film taken March 23, 1954, eleven years and three months following the bone graft, shows essentially the same findings as in the previous print. Note the clear open medullary canal.



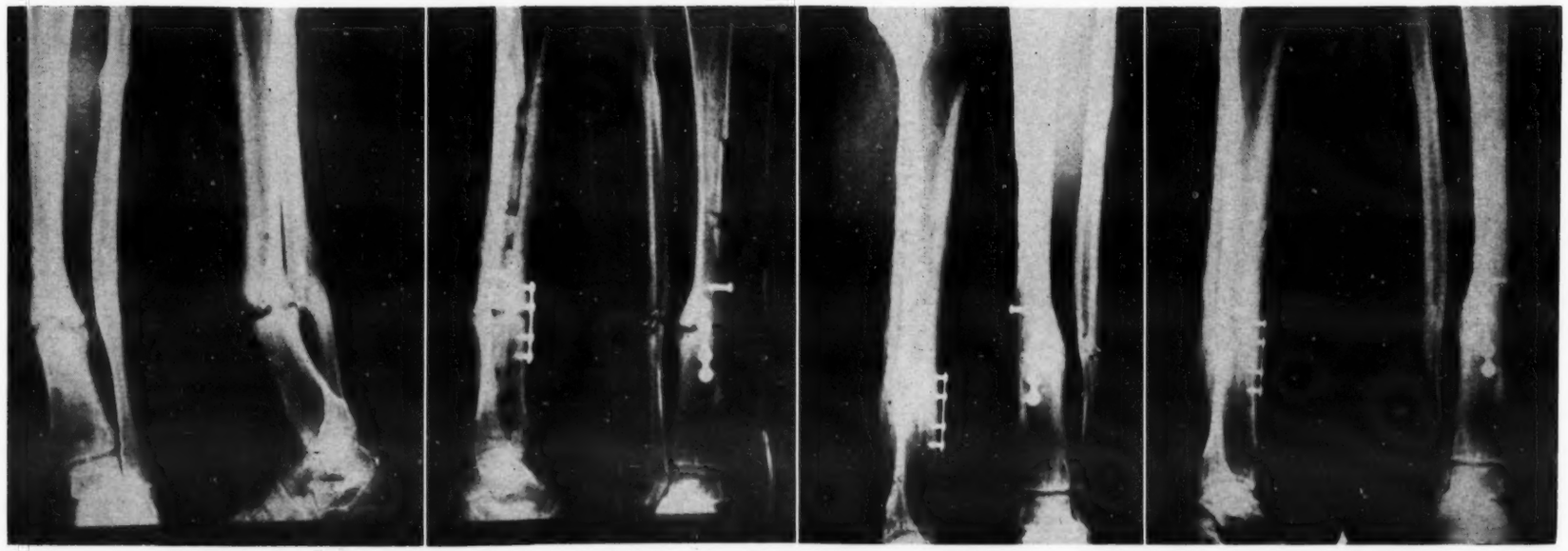
4A 4B 4C 4D

FIG. 4. Case III. A, film taken December 11, 1943, one month prior to grafting. Note the absorption of bone at the fracture site along with fracture of the bone plate and some osteoporosis.

B, film taken January 6, 1944, two days following the bone grafting procedure. Note the dovetail at the distal end of the major graft. This graft is held in place with two screws above the fracture site, and the minor graft is transfixed with a single Sherman screw.

C, film dated March 11, 1944, two months following bone graft. This shows that the grafts had healed nicely to the major tibial fragments and to each other. Note that the fracture line is almost completely obliterated, even so short a time following the operative procedure.

D, film dated June 7, 1955, eleven and one-half years following the bone grafting procedure. Note the normal appearance of the tibia so far as shape is concerned. Neither the graft sites nor the original fracture site can be clearly identified. There is no absorption around any of the screws, the medullary canal resembles that in a normal bone, and except for the presence of the screws one would hardly know that this bone had been operated upon.



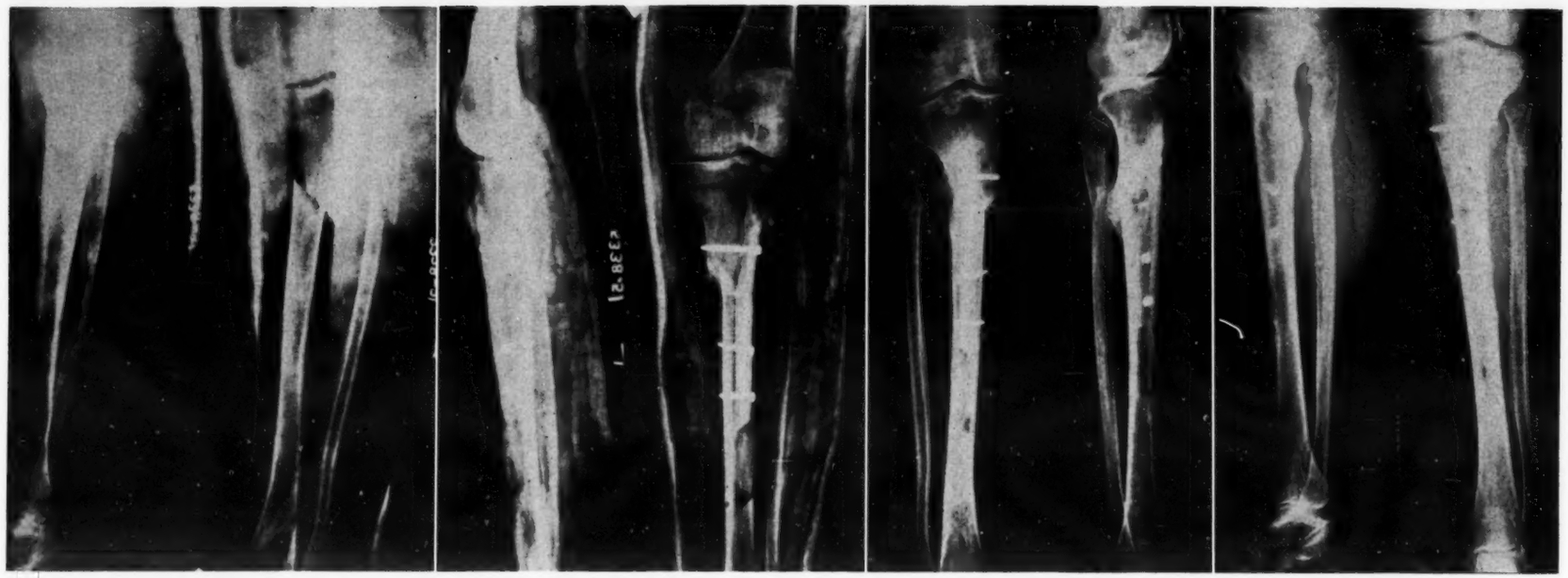
5A 5B 5C 5D

FIG. 5. Case IV. A, film taken August 18, 1943, five days prior to operation. The patient had been walking without external fixation for over a month and had received a series of paravertebral blocks. In addition, the draining sinus had been excised and the skin was well healed. This film shows the malunited fibular fracture, the ununited tibial fracture with sequestra at the fracture site, and marked posterior angulation of the distal tibial fragment.

B, film dated August 24, 1943, the day following operation. Note the fibular osteotomy which was necessary to correct mal-alignment and posterior angulation. Note also the Sherman plate which was necessary to maintain alignment of the tibial fragments. The lateral graft is fixed with a single Sherman screw above the fracture site, and the distal end is well held by the dovetail technic. Note the presence of three smaller grafts above the major graft.

C, film taken March 4, 1944, six and a half months following the bone grafting procedure. Note that all grafts have blended into the major tibial fragments and have healed to each other. There is good union of both tibia and fibula.

D, film taken January 10, 1954, eleven years and five months postoperatively. Note complete bony healing of both bones with open medullary canals and good alignment. One cannot tell that this tibia has been grafted. Appearance here is one which resembles a healed fracture which was treated by open reduction with plate and transfixation screw type of immobilization.



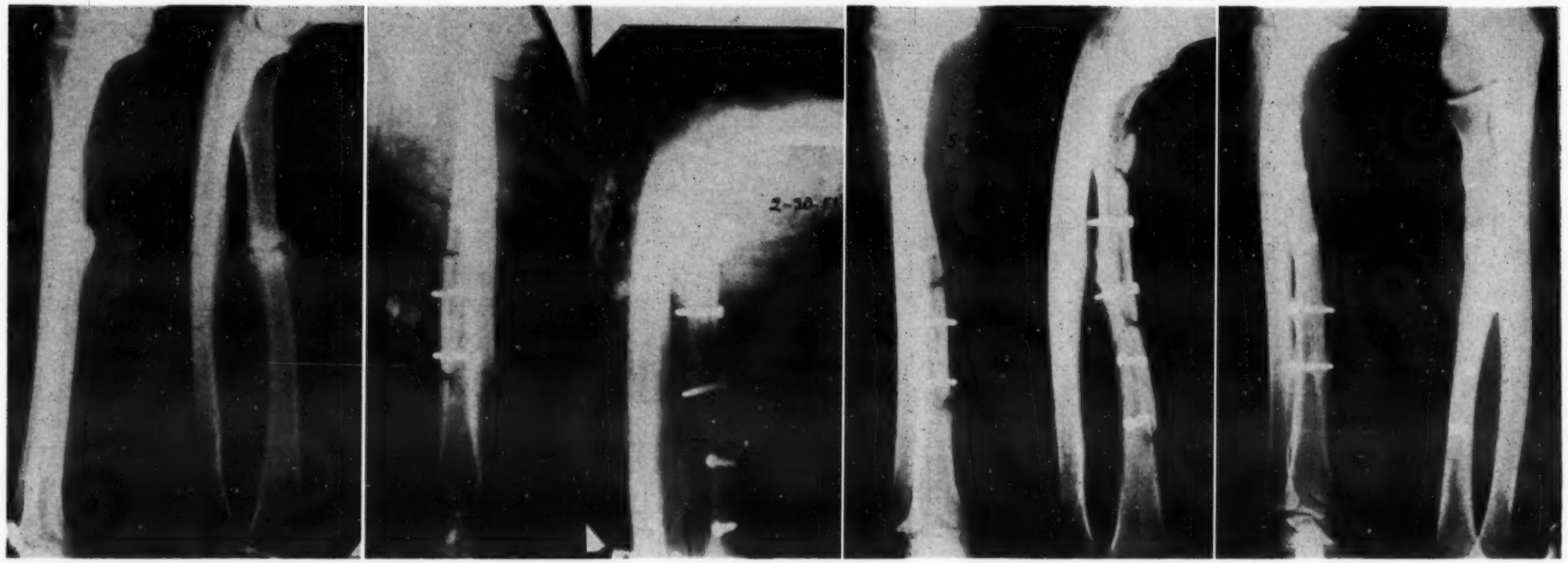
6A 6B 6C 6D

FIG. 6. Case v. A, film dated June 3, 1951, two days prior to operation. Note the displacement medially of the distal tibial and fibular fragments with no evidence of healing.

B, film taken June 7, 1951, shows the immediate postoperative views. Note that the mal-alignment of the tibia has been corrected and that the distal tibial fragment has been impacted into the upper fragment and that the major graft has been well pushed into the tibial plateau. The upper transfixation screw was used to take the tension off the graft at the fracture site. Note the gaping area at the distal end of the donor site.

C, film taken October 7, 1951, four months following the grafting procedure. Note that almost complete bony union has taken place and that the graft is barely visible. Note, too, that the gaping area at the distal end of the donor site has filled in and that the medullary canal is open.

D, film taken April 8, 1954, two years and ten months following the grafting procedure. There is excellent bony union, and the bone graft can barely be differentiated from the tibia proper. Note that the medullary canal is open and that the appearance of the tibia so far as shape and contour are concerned almost resembles the normal.



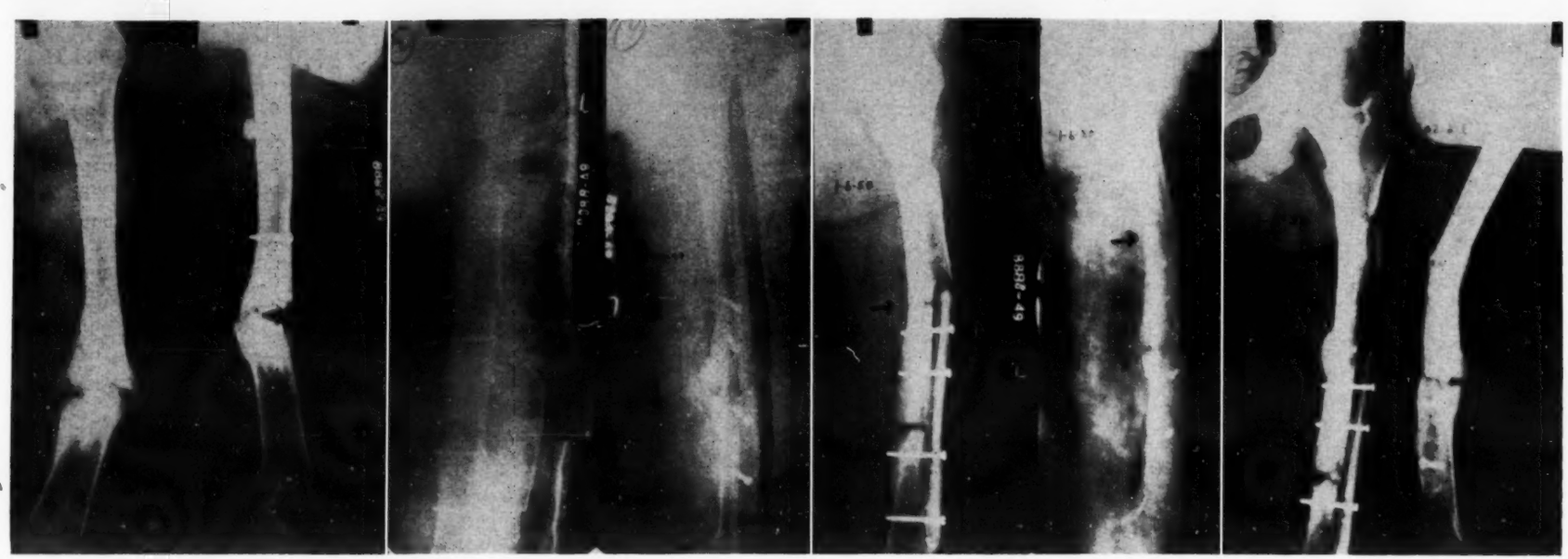
7A 7B 7C 7D

FIG. 7. Case VI. A, film taken January 15, 1951, one month prior to operation. This shows the ununited fracture of the radius with excess callus in the subperiosteal area.

B, film dated February 20, 1951, the day following operation. Note that the larger graft is not much different in size from the smaller, this resulting from the accidental fracture of the major graft. At the upper end note the small bone fragment which is a broken-off portion of the graft and which has been placed loosely in the intramedullary area.

C, film dated May 14, 1951, three months following the operation. Note that all three graft fragments are blending into the major bone fragments and that some healing of the ununited fracture is taking place.

D, film taken May 17, 1955, four years and three months postoperatively. There is complete bony healing with a normal appearing radius and an open medullary canal. Note that it is difficult to identify the small broken-off fragment of the graft.



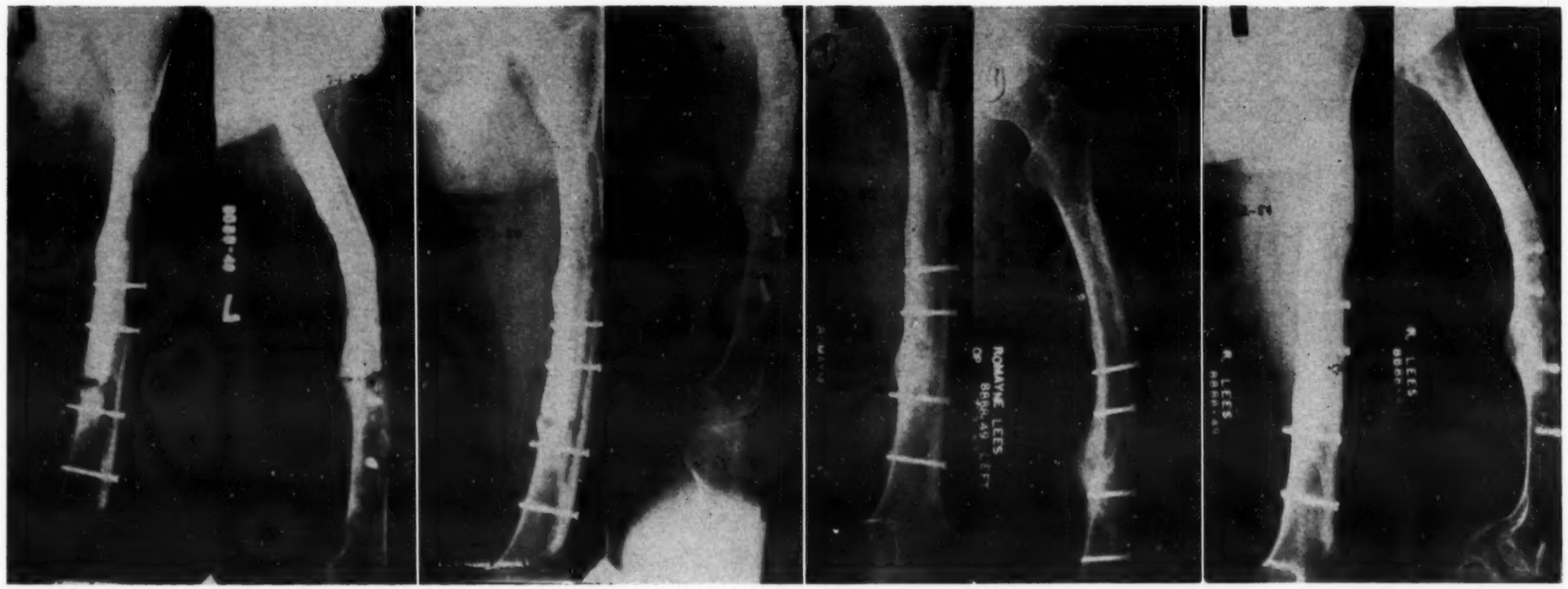
8A 8B 8C 8D

FIG. 8A to D. Case VII. A, film taken on admission to the hospital on September 9, 1949. Note the absorption of bone at the fracture site along with the remnants of the previously inserted intramedullary bone graft as well as the rounded off and completely closed intramedullary canal at the site of the ununited fracture.

B, film taken September 15, 1949, shows the major graft held by four Sherman screws. There was no internal fixation for the smaller graft.

C, film taken one month later on January 6, 1950. The patient had slipped over the New Year's holiday and had fractured the shaft of the femur at the upper level of the major graft. Note that the graft is healing into the major femoral fractures and that it is intact at the site of the ununited fracture.

D, film taken on March 6, 1950, eight weeks following the fracture of the femoral shaft. Note complete bony healing of the fracture site and the excess callus along the medial aspect of the shaft of the femur. Note, too, that the upper graft was completely healed to the femur and that incomplete healing is present along the major graft. The only treatment for this shaft fracture was plaster immobilization.



8E 8F 8G 8H

FIG. 8E to H. Case VII. E, film dated July 1, 1950, ten months following the grafting procedure. The shaft fracture is completely healed, the grafts healed into the major femoral fractures, but the site of the ununited fracture is still unhealed.

F, film taken September 2, 1950, one year following the operation. At this time the patient was in for a routine check x-ray. When the films were examined, the fracture of the graft proper at the site of the ununited fracture was noted. The patient was not complaining of any discomfort and was surprised to learn of this finding. Treatment consisted of plaster immobilization.

G, film dated July 3, 1952, two years and ten months postoperatively. Note the open medullary canal, the complete healing of all the previous fracture lines and the fairly normal anatomic contour of the femur.

H, film taken April 22, 1954, four and a half years postoperatively. There is complete healing with an open medullary canal and with but slight increase in the normal anterior bow of the femur. There is no reaction about any of the screws.

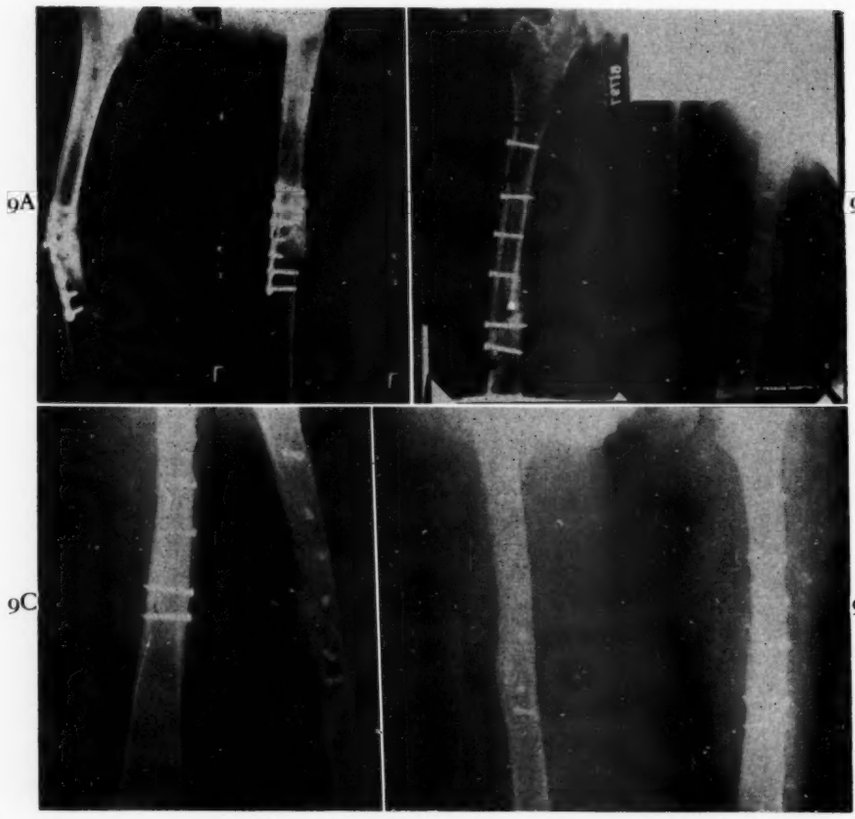


FIG. 9. Case VIII. A, film taken October 14, 1941, four months prior to operation. Note the marked angulation and the fracture of the bone plate along with absorption around the transfixation screw. B, film taken March 2, 1942, several days following the operative procedure. Note that the massive major graft is immobilized with five Sherman screws which were necessary to overcome the pull exerted by the muscles. The smaller fragment is fixed with a single screw. Note, too, that the ends of the graft are not dovetailed but are cut straight across. C, film taken December 7, 1945, three years and nine months after the operative procedure. Note that excellent bony union has taken place at the site of the ununited fracture, that the medullary canal is open, and that there is no absorption around any of the screws. The graft sites cannot be identified at this time. D, film taken May 16, 1955, thirteen years and two and a half months after operation. There is no essential change from the previous print and the femur has a normal shape and appearance. There is still no reaction around any of the screws.

is the best type of bone grafting procedure for the treatment of ununited fractures of long bones.

The most important point we wish to bring out is the fact that autogenous bone is much better than heterogenous or even homogenous bone for grafting purposes. The patient's own bone is always superior to that obtained from a bone bank, and we might add that bone taken from the bone involved in the ununited fracture is superior to bone taken from any other part of the patient's own body. Because of this we believe that the procedure is of value, for it eliminates the necessity of the bone bank as well as the need for a second operative site to carry out a bone grafting procedure. In every instance, at the time of the first bone grafting operation, we have always been able to obtain sufficient bone for grafting purposes from the involved bone itself. In some cases a second and even a third grafting procedure has been carried out without resorting to another bone or the bone bank for a graft.

Another point of interest is that all of these cases show the importance of an open intramedullary canal. In every patient with un-

united fracture that we have operated upon, we have found the fractured ends of the bone at the ununited site to be rounded off and the medullary canal sealed over with either dense fibrous tissue or bone itself. This is true of patients who have or are without calcific deposits in the surrounding soft tissue. With this avascularity it is impossible for the fracture to heal, and because of this we wish to stress the importance of cleaning out the site of the ununited fracture and of creating and maintaining an open medullary canal above, below and across the site of the fracture. We believe that the cases presented herein bring out this point clearly, for one of the first things that nature does, as healing takes place following this grafting procedure, is to establish a new open medullary canal.

Of interest, too, is the type of healing which takes place between the major bone fragments and the grafts, and between the grafts themselves. It has been noted that these grafts heal by blending into the major bone fragments above and below the traversed fracture site. By immobilizing the graft tightly into the major fragments of the involved bone, one is able to set up osteogenesis and healing in a manner which can, in principle, be compared with the type of healing which takes place when a comminuted fracture is treated by open reduction and internal fixation. Bony healing takes place across the multiple fracture sites with little or no subperiosteal callous formation. At the same time, the osteogenetic process set up by the fresh marrow across the site of the ununited fracture stimulates healing and bone formation at this area and brings about the production of enough new bone to bridge the ununited fracture and to blend this in with the bone graft proper. Where the bone grafts do not come in contact with each other or where there is a gaping area above as well as below the lesser graft, the same type of healing reaction is set up, and over a period of four to twelve months nature has completely filled in these areas so that, as an end result, one obtains a bone which is of almost normal shape and contour. There are no bulky deformities, there is no bulbous callous, very seldom is there fusiform subperiosteal callous formation at the fracture site, and in most instances a year or two following the operation one cannot tell that the bone has been grafted. In fact, in all of these cases the final result looks more like a bone which has had a simple reduction of a fracture with some type of internal fixation. These changes take place within the first year or two, and following this period very little if any changes are noted in the radiographic appearance of the bones. We believe we can safely make the statement that after a year and a half to two years have passed anyone examining an x-ray film of one of these bones could not tell that the bone has been grafted. Even when one has seen many of these patients postoperatively and has then seen a long-term follow-up, it is difficult to discern the areas where the grafts were obtained and placed without careful study of the picture. We believe that our follow-up periods in these cases have been of sufficient length and over a large enough series of cases to warrant our safely making the aforementioned statements.

Also of importance are the technical difficulties encountered because of muscle pull and malposition of these limbs over a long period of time. It is difficult for one to determine except at the operating table the type or amount of internal fixation which is required for that individual. This can be answered only by the operator's individual experience. One thing to remember, however, is to use enough internal fixation to completely immobilize the major graft, for motion of this graft, especially across the site of ununited fracture, could easily cause it to break. In addition, constant motion would prevent good healing from taking place.

There are certain complications which can and do arise, and one must keep in mind the possibility of their occurrence and what to do for them when they present themselves. The graft can fracture across the site of the ununited fracture. The shaft can, and at times does, fracture through the donor site above or below the site of the ununited fracture. Infection which often has been present in compound cases may be stirred up and cause a long postoperative period with failure of the graft to heal. This has happened to us in two cases, both within the past two years. These complications will not be discussed in detail at this time but a later presentation will cover the manner in which these complications were and can be handled. An attempt at that time will be made to present and analyze the thirteen patients who required more than one operation and the patients in whom infection developed during the past year.

CONCLUSION

A type of bone grafting procedure which has been in use for thirty-three years and which has given us excellent results in a large series of cases has been presented. We have reported on patients with follow-ups of almost twelve years and have analyzed 207 bone grafts done on 182 patients in the course of 196 operations. We believe that this procedure is superior to any of the other types of bone grafting procedures commonly used. The results speak for themselves.

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Unusual Farm Machinery Injuries

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From the Hatch Clinic, Idaho Falls, Idaho.

Since a large part of our patients come from rural areas, we have selected three unusual farm machinery injuries for presentation.

Case 1. This patient was a thirty-eight year old farmer who was admitted because of complete avulsion of the skin of the genitalia. His clothes were caught in a potato digger power take-off; they were twisted and torn completely off, leaving only shoes. Even the socks were torn out of the man's shoes. The skin of the scrotum and penis was caught and pulled off with the clothes. (Fig. 1.)

He was taken to surgery where the denuded area was irrigated, scrubbed and débrided. A split-thickness graft from the thigh was used to cover the penis. The testes were implanted under the adjacent skin. A small remaining scrotal tag was sutured back in place. It later became necrotic and was replaced by a second skin graft. At the time of the second operation the shaft of the penis appeared shortened and was lengthened with a portion of the same new skin graft. The final result, as illustrated in Figure 2, shows good coverage with the skin softening satisfactorily.

This man has three children. Since he has no desire for more, sterility is not a problem. Sexual function is satisfactory. He has no pain or testicular compression with any movement. His only complaint, when last seen, was the possible desire for circumcision since the grafted skin did not seem to retract easily.

Comment. This injury, although rare in the literature, is becoming more common. In 1950 Gay and Oates[1] reviewed fifty-three cases. A few cases have been reported since that time. Many authors have contributed technics of reconstruction. The summaries of Owens[2] in 1942, Ewell[3] in 1953 and reports of others[4–6] have tended to clarify present-day principles of treatment.

Most authors agree that the best covering for the denuded penis is a relatively thick split graft from a hairless area. A single graft is desirable to cover the entire penile shaft. The suture line placed along the dorsum in a zig-zag line will minimize subsequent scarring and limitation of erection. Removal of any skin remaining attached to the corona is recommended because it frequently swells and becomes necrotic if left attached.

Fig. 1. Case 1. Preoperative view showing avulsion of scrotal and penile skin.

Fig. 2. Same case. Picture taken two years postoperatively. All of the skin covering is pliable.

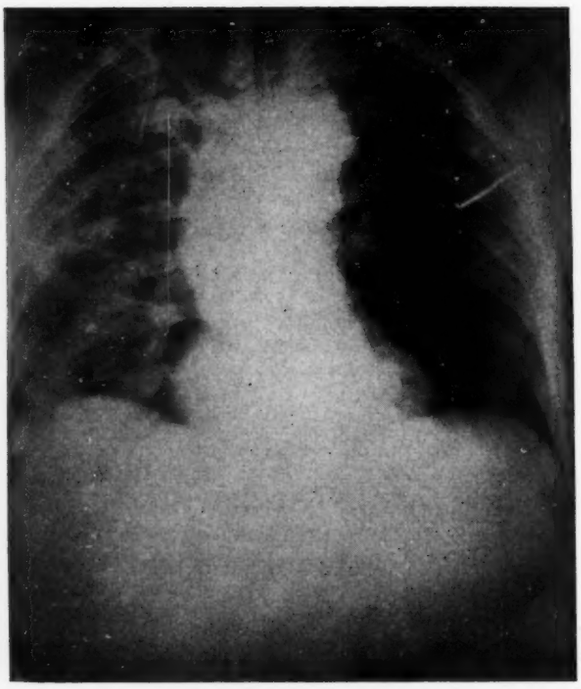


FIG. 3. Case II. Chest x-ray on admission of patient; film showed foreign metallic body high in left chest, slight diffuse clouding of right lung field and a large shadow in the mediastinum.

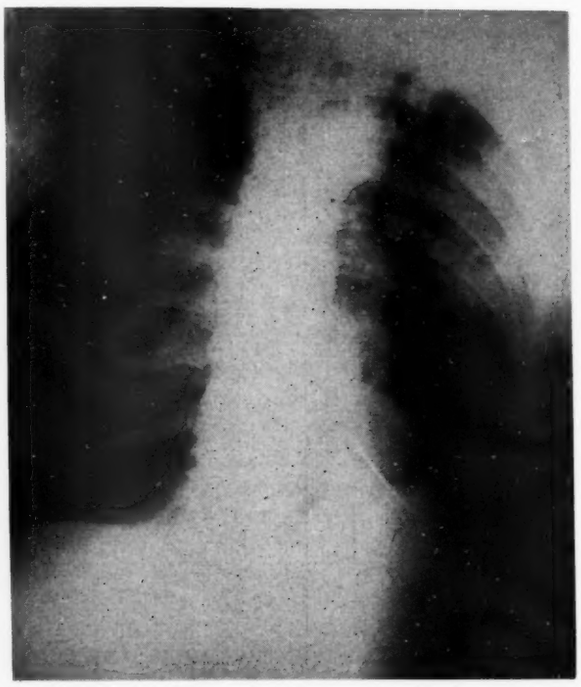


FIG. 4. Same case. Several hours after admission chest x-ray shows collapsed right lung. The foreign body has dropped to a lower level.

There is considerable difference of opinion regarding scrotal reconstruction. Satisfactory results have been obtained by transplantation of the testes under adjacent skin, as in the present case. Also, scrotoplasty may be carried out in a single stage or in multiple stages with pedicle grafts. There is doubt that reconstruction of a satisfactory thermoregulatory scrotum is possible.[5] Therefore the prospect of maintaining spermatogenesis by any known method is uncertain. However, it would seem that a sac further removed from the body heat would offer the best hope.

CASE II. This case involved a freak farming accident. A fifty-two year old farmer was brought to the hospital emergency room with a history of having been hit in the back with a stone. He had been driving a tractor, pulling a sagebrush clearer.

The farmer was short of breath and complained of pain in the chest. Examination disclosed some subcutaneous emphysema over the right chest. No definite rib injury was found. The lung sounds were fairly clear. X-ray of the chest showed a metallic foreign body high in the left chest, slight diffuse clouding of the right lung field and a large shadow in the mediastinum which was diagnosed by the radiologist as an aneurysm. (Fig. 3.)

Re-examination of the patient disclosed a pinpoint puncture mark over the posterior right seventh interspace. A 3 inch piece of fence wire, thrown with great force, had traversed the right posterior chest wall, the right lung and the mediastinum, and had lodged in the left pleural sac. Bleeding into the mediastinum gave an x-ray shadow simulating aneurysm.

Several hours after admission total right pneumothorax had developed. X-ray at this time showed that the right lung had collapsed. The foreign body in the left chest had dropped to a lower level. (Fig. 4.)

By means of a needle water seal, the right lung was re-expanded. However, extreme subcutaneous emphysema developed from the cheeks down to the knees and hands. The patient became semi-comatose and remained so for over forty-eight hours. Thereafter the emphysema gradually disappeared. The patient was discharged much improved on the tenth day.

Three weeks later he was readmitted in good condition and was given a therapeutic left pneumothorax of 1,000 cc. A urinary panendoscope was passed through a small stab wound into the left chest. The wire was identified in a fresh adhesion, grasped with a stone forceps and removed. The patient's subsequent course was uneventful. A final x-ray showed complete resolution of the mediastinal shadow. (Figs. 5 and 6.)

FIG. 5. Case II. Picture illustrating removal of wire from left chest with panendoscope.

FIG. 6. Same case. Final x-ray showing complete resolution of mediastinal shadow five weeks after date of first admission.

Comment. Here is an instance of a piece of wire being thrown at such great velocity that it penetrated the right side of the chest from behind. It traversed the right lung, the mediastinum, and lodged in the left pleural space. A right pneumothorax and marked subcutaneous emphysema developed, as well as bleeding into the mediastinum which simulated an aneurysm. All of these abnormalities cleared and the foreign body was removed from the left chest by means of a urinary panendoscope and an alligator stone forceps.

CASE III. This patient was a three year old boy who had been run over by a disc harrow. His head was wedged between two of the discs. The remaining discs ran over his neck, chest, arms and legs. The soft soil cushioned him enough so that no bones were broken. However, the anterior part of the neck was flattened and he was neither conscious nor breathing.

It took the father's full strength to free the boy's head from between the blades. According to the father, during the rough truck ride to the hospital the neck popped back out and then breathing resumed.

Initial examination revealed an unconscious boy with dilated, equal pupils, labored respirations, heavy moist rales on both sides and disc marks

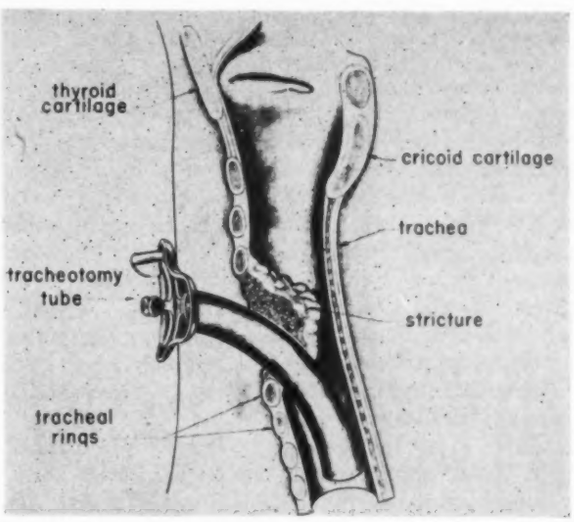


FIG. 7. Case III. Shows the tracheotomy tube in place with the stenosis above due to scar and granulation tissue.

across the neck, chest, abdomen and legs. Oxygen and suction restored consciousness.

At first intratracheal aspiration served to remove secretions. However, on the third morning increased secretions and labored respirations necessitated emergency tracheotomy. While the boy was being prepared for this, he stopped breathing.

When the neck was opened, a contused, lacerated trachea was found and large amounts of fluid welled

FIG. 8A. Case III. X-ray showing rubber tube in trachea spanning the scarred area; the tracheotomy tube enters through the distal portion of the rubber tube.

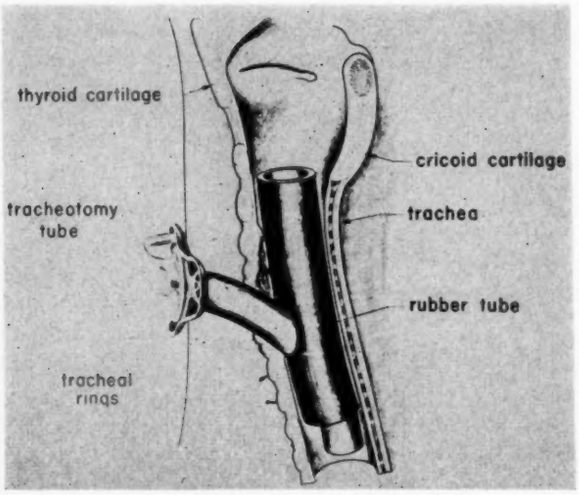


FIG. 8B. Diagram of Figure 8A.

out of the wound. Prompt suction, insertion of a tracheotomy tube and five minutes' artificial respiration were required to restore breathing.

An attempt was made to repair the trachea above the tube. Pieces of cartilaginous tissue and loose friable material were found but there was no good anterior support in the area of the third and fourth rings. The posterior tracheal wall was intact.

The tracheal injury presented a most difficult problem because stenosis due to scar and granulations developed above the tracheotomy. (Fig. 7.)

In searching for methods of repair, three unsuccessful technics were tried:

First, three weeks after tracheotomy a rubber tube splint[6] was inserted. Figures 8A and B show the rubber tube spanning the scarred area with the tracheotomy tube extending through its distal portion. With this tube in place the tracheotomy tube could be plugged and the child could cry and breathe fairly well. After three more weeks, when the rubber tube was removed, the boy was still unable to breathe sufficiently and the tracheotomy tube had to be replaced.

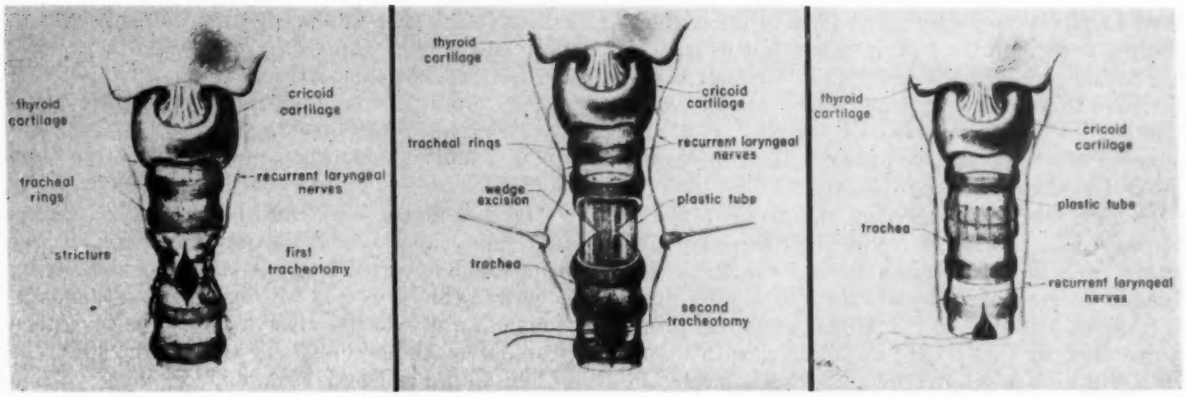
Second, retrograde dilatation of the scar tissue was done using the obturators of tracheotomy tubes in graded sizes. This was done every morning for three weeks but was unsuccessful because the lumen grew back with granulation tissue and scar as rapidly as it was dilated.

Third, approximately nine weeks after tracheotomy, using a conical endocervical biopsy instrument, the scar and granulation tissues were excised from the area of stricture and the rubber tube and tracheotomy tube reinserted. The patient could breathe well and talk with the tracheotomy tube plugged and was able to have it plugged continuously. He was discharged to his home and followed up until twenty weeks after tracheotomy. During this time he required occasional suction but was cared for by his parents. He then began to have additional respiratory distress. It was thought that granulation tissue was closing off the trachea above the rubber tube.

Therefore, five months after injury, a new tracheotomy was performed lower down, at the sixth to seventh ring level. (Fig. 9B.) The old wound was excised down to the trachea. Both recurrent laryngeal nerves were identified low in the neck and carefully freed from the scar tissues up to their entrance into the larynx.

The original tracheotomy opening with its scar and granulation tissue edges and the stricture above were excised in a large wedge back to healthy tissue. This left a narrow segment of posterior trachea intact and a defect of about 1 inch anteriorly in the area of the third and fourth rings. A short polyethylene tube was placed snug in the lumen; the trachea was remarkably mobile and was closed end-to-end over the tube by bringing the second and fifth rings together with interrupted stainless steel wire. This effected a neat, air-tight closure with only slight buckling posteriorly. (Fig. 9C.)

A silk suture attached to the lower end of the polyethylene tube was brought out through the new tracheotomy opening and used to remove the tube four weeks later. Bronchoscopy at this time showed a well healed trachea with no evidence of



9A 9B 9C

FIG. 9. A, diagram shows the tracheal stricture with the tracheotomy opening. Laryngeal nerves are bound to the scar tissue. B, shows a new tracheotomy at the sixth to seventh ring level; recurrent laryngeal nerves freed from the scar tissues. Wedge excision of the area of scarring and a polyethylene tube splint in the trachea with a silk suture attached to the lower end and brought out through the new tracheotomy. C, shows end-to-end closure over the plastic tube bringing the second and fifth rings together with stainless steel sutures.

stricture. The tracheotomy tube was removed a week later and the wound healed promptly. It has now been a year and a half since operation and the patient has had no further tracheal symptoms.

Comment. Rupture of the trachea without lateration of the skin may occur with blunt trauma. It is interesting that the patient may be able to breathe reasonably well for periods of many hours. In some cases subcutaneous emphysema may not follow.

Dysart[9] reported a case of rupture and complete separation of the trachea from the larynx with no subcutaneous emphysema. His patient was able to breathe fairly normally for fifteen hours but then required tracheotomy. Subsequent stenosis was treated successfully by endotracheal excision of the scar, followed with rubber tube splinting for six months.

Erich[10] of the Mayo Clinic reported successful treatment of tracheal and laryngeal stenosis by excision of the scar tissue and lining the area of defect with a split-thickness skin graft around a rubber sponge.

The present case illustrates a method of repair by which a wedge section of the scarred trachea was excised. The defect was closed by mobilizing the adjacent edges and suturing them end-to-end over a plastic tube. Neither prolonged splinting nor later dilatations were necessary.

CONCLUSIONS

Three cases of unusual farm machinery injury have been summarized. The results of their treatment have been reported along with comments about treatment gleaned from similar reported cases.

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DISCUSSION OF PAPERS BY DRS. TODD, WAGNER AND FERRARO, AND HATCH AND JONES

MARTIN C. LINDEM (Salt Lake City, Utah): I want to congratulate Drs. Hatch and Jones for

their excellent presentation on farm injuries. I also want to commend Dr. Hatch for promptly responding to his privilege and duty to be articulate and active in this society, to which he has recently been elected. The latent period of activity of a new member is too frequently manifest, and the example of Dr. Hatch is to be followed.

Farm injuries are primarily the problem of the country doctor, and too frequently there is inadequate care. The big hospitals and clinics do not assume immediate responsibility and are rarely first called to see farm casualties. Thus this group is attended by inadequate initial care, with immediate disastrous results or ultimate unnecessary disabilities.

I hope this paper will emphasize an object for this society, that is, to consider farm injuries on the same serious level as the highway and automobile injuries, transportation injuries.

A prevention and safety level is involved. The National Safety Council has called attention to the predominance of farm injuries.

Dr. Howard Snyder of our own group has been a persistent orator on the handling of this type of injury. Farm injuries are necessarily severe because of the heavy equipment employed and the relative ever-presence of inexperienced operators of the machinery. The incipiency of tetanus and gas bacillus infection is incident because of the presence of farm animals which carry the seeds of this infection.

The industry of farming deserves that we place at its disposal safety measures, training of surgeons and prevention instruction comparable to that given heavy industries who compete for privilege on safety programs. This should be a project of the surgery of trauma.

Dr. Hatch's paper shows that the surgeon in the small town has versatile skills. I must say that Dr. Hatch is not exactly from a small town because Idaho Falls is rather large, and the excellent Hatch Clinic is the number one project. However, he also shares the farm population as patients.

You will note that he is able to use a cystoscope to remove a foreign body from the thorax, and he shows the versatility of the thoracic surgeon in handling the trachea.

It would be well that our young surgeons could be trained in this versatility when they go to the smaller towns because they will be called upon to use this skill.

The farmers deserve our attentive consideration of their needs just as do the automotive casualties and those injured by burns and in industry.

Dr. Hatch, I hope you will persevere in this field of surgery and reporting. Thank you for an important paper.

Walter Scott Nettrour (Pittsburgh, Pa.): I wish to differ mildly with Dr. Todd on perhaps a minor point, that is, the question of Army casualties versus Navy casualties.

Inasmuch as the Army frequently has air evacuation even with helicopters and inasmuch as the Navy frequently has amphibious landings with soldiers and sailors, the end result is often very similar.

I wish to state, also, in Dr. Ferguson's absence, that many of his casualties on the U.S.S. Solace from which he wrote his paper were from the Army as well as the Navy and Marines. Also, ideal conditions naturally do not always exist. The seventeen year old machine-gunner on the aftermast of the U.S.S. San Francisco remained on duty with a compound fractured femur for three days with no light, no heat and cold rations; he was taken over the side after three days, when there was not even enough material to make a proper splint for the fracture.

I do believe that Dr. Ferguson realized early the value of antibiotics; also, he made the point that many men here have made in their papers, i.e., that débridement certainly can be overdone.

I wish to congratulate Drs. Ferraro and Wagner on an excellent paper on compound fractures.

Harrison L. McLaughlin (New York, N. Y.): The paper by Drs. Wagner and Ferraro is a study of results in a group of long bone non-unions treated by sliding grafts. In terms of quantity, quality and detailed long-term follow-up it is a magnificent report. The excellence of the results bespeaks the surgical technic of the authors sufficiently to make further comment along this line superfluous. Drs. Wagner and Ferraro are to be congratulated. However, I think there is some room for discussion concerning the factors advanced as reasons for such good results.

The report credits the sliding inlay graft with certain advantages:

1. *One can always get enough bone from the host area.* This is usually true, but is it not fraught with some difficulty and danger in the forearm? Did not three of fifteen (20 per cent) of the grafted forearms fail to unite after the first operation?

2. *There is no need for a bone bank.* This may be true, but in the days of direct transfusions it was often thought that there was little need for a blood bank.

3. *The inlay graft opens the sealed-off medullary canal.* Is not a successful graft of any type eventually attended by reconstitution of both the cortex and canal of the bone?

Several of the principles postulated deserve discussion:

1. *The medullary canal should always be opened and the pseudarthrosis excised.* There is, of course, general agreement with this, except for the word "always." Under certain conditions the pseudarthrosis is better left undisturbed; but when it is

excised, it would seem appropriate to eliminate the residual gap and secure apposition of fragment to fragment as well as graft to fragments, even at the expense of cutting the fibula.

2. *Osteoporosis contraindicates grafting—the medullary circulation is of paramount importance in healing.* But, osteoporosis is synonymous with hyperemia. I cannot agree that osteoporosis in itself contraindicates grafting, except when it constitutes a red flag indicative of decreased vascular and lymphatic efficiency throughout the limb.

3. *A graft from the host bone is better than one removed elsewhere in the body.* Is this not the equivalent of saying a slightly sick bone graft is better than one from a normal bone?

Finally: *An autogenous graft is better than a homogenous graft.* It certainly is fresher, but no less dead; and despite postulations to the contrary, so long as the preserving process does not alter the protein matrix of a graft, I have not observed any clinically demonstrable difference between the two. We now sterilize our bank bone by irradiation; this method promises to be the safest and most convenient technic yet devised.

G. W. N. Eggers (Galveston, Tex.): I think the greatest contribution to the non-union problem is its prevention. I believe that this prevention can be accomplished by following certain principles in treating the patients operated upon in open reduction at the primary operation.

This is to be done by following the principle of the three C's: the principle of creating continuity, contact and compression. I believe that when many of the cases presented are analyzed, they show that the three C's were applied in the final analysis.

In treating 237 closed cases by open reduction and following the principles enunciated, non-union occurred in two cases, which is less than 1 per cent of our cases.

I do not think that the use of the sliding grafts in the round bones of the femur and forearm is particularly desirable. As far as the use of grafts is concerned, it is my belief that each person has a "bone bank" in his ilium from which satisfactory grafts can be secured.

I am going to state again that the adoption and use of the three C's (continuity, compression and contact) in the treatment of fractures of any sort will lead to a high rate of union.

Oscar Perry Hampton, Jr. (St. Louis, Mo.): Since Howard Snyder did not challenge Dr. Todd, I feel that I must do so. The most important point in the prevention of the septic compound fracture, in my opinion, is adequate débridement.

Débridement is a precise surgical procedure. It does not mean radical block excision of tissue. It does mean the removal of all contaminated, devitalized tissue. Some open fractures may have a minimum of devitalized tissue, and if so, perhaps thorough irrigation and a little trimming remove all of the devitalized tissue. However, I cannot accept the concept of Dr. Todd that all that counts is the degree of contamination as it varies between the open fracture produced by a shell fragment passing through a sailor's clean clothes against the mud and grime of battlefield, etc. It is the destroyed tissue in the wound which creates the pabulum for the septic process which will develop in a large number of open fractures if adequate débridement is not performed.

M. H. Todd (closing): It is well that during the reading of this paper I repeated several times the fact that I was using my imagination. I notice the Navy objected to my thinking that all naval wounds are clean and that they always get prompt treatment, and I certainly cannot question this.

F. P. Ferraro (closing): I would like to thank Dr. McLaughlin for his analysis and criticism of our paper. There is no way to learn more than to have somebody criticize him.

I will try to answer the questions which he brought up. The first was in regard to the forearm. In the cases in which the grafts failed, failure was not due to lack of bone for the graft but was due to mechanical reasons. Let me say that in these cases there was ample bone for each and every graft. The failures were three in number, and of these, two grafts failed to heal. In the other, the graft healed but a fracture of the graft at the site of the ununited fracture caused the failure. Dr. McLaughlin mentioned a 20 per cent failure in forearm grafting. I beg to disagree with him, as our figures show three failures in ten cases when both bones of the forearm were grafted at the same time, one failure in fifteen cases involving the radius alone, and one in fourteen involving the ulna alone. This makes five failures of forty-nine grafts, or approximately a 10 per cent failure. This we believe is not bad for forearm grafting.

As to the medullary canal, we have opened it in all instances and still beg to disagree with Dr. McLaughlin in that we believe intramedullary healing is important. We use as our basis for this statement the radiographic evidence noted during healing of long bone fractures which have been treated by intramedullary pinning. Here, all of us who have used this procedure know that healing takes place subperiosteally with no intramedullary healing. The fracture line remains visible for a long time and is last to disappear, even though the patient has been walking on the leg for a long period of time. We still think that we have the advantage of healing both from within and without.

As to the approximation of the major fragments during the grafting procedure, we attempt to do so in every case in which it is possible. When there is a major defect, malposition or malalignment, we do not hesitate to perform fibular osteotomy in a

tibial ununited fracture. The one case which we presented brings out this very point. In other cases when the defect is not too great, we rely on the graft to bridge the defect and leave the fibula intact as we believe that it has a steadying influence in helping to maintain immobilization.

On the point of osteoporosis, Dr. McLaughlin states that osteoporosis is hyperemia. This may be so, but soft mushy bone, so far as we are concerned, does not seem to give an ample graft. We have seen men attempt this, and it is in these cases that the percentage of failures is high. One question arises in my mind: "Is this hyperemia a true hyperemia or is it stagnation of blood in the involved bone due to its disuse demineralization and poor circulation"?

In regard to autogenous bone verses bone from the bone bank, we still believe that if you can obtain bone from the involved area there is no need for the bone bank. We have nothing against bank bone, but we still prefer to use a graft from the immediate area if it is available. As to using bone from another area in the same individual, we prefer our method as we are operating on but one bone, carrying out one operation, and subjecting the patient to the shorter operating time. Why perform two operations and prolong the patient's stay in an anesthetized state when a single procedure will suffice?

One other point. As the discussion covered the field of antibiotics, I have a comment to add. We are not believers in the prophylactic use of antibiotics, and in the greatest number of our cases no antibiotics were used. Their use was restricted to patients who had infection or draining sinuses at the time when we first saw them.

In regard to open reduction on the twelve year old boy, may I state that this is not our routine treatment. We attempt closed reductions in all such cases and resort to operative procedures only when we are dissatisfied with the results of the closed reduction. Even though Nature will heal most of these fractures regardless of the position of the fragments, we still like to give these children the benefit of an anatomic reduction and will operate upon them if we cannot obtain a desired reduction by other means.

Treatment of Mass Mechanical Injuries

COL. JOSEPH R. SHAEFFER, M.C., *Washington, D. C.*

THE treatment of traumatic injuries is a continuing problem in the fields of both civilian and military medicine. The human body is readily injured by violence, a recurring characteristic of its environment. Man constantly seeks means of protection from trauma and better methods of combating its effects. Accidental injuries are the fourth most common cause of death in this country; over 10,000,000 are recorded each year. There can be little doubt that many times this number are unreported.

In recent years several areas of our country have suffered sudden disaster of natural origin, each accounting for loss of many lives and many injuries requiring professional care. In our time this nation has been involved in three major wars, each with its toll of death and wounded produced by man-made devices. In each instance our medical resources have been mobilized to care for these casualties. Furthermore, the uncertainty of the future demands that all those joining our profession in the time to come participate in our military preparedness program. Our educational institutions and postgraduate training programs are becoming increasingly alert to the need for greater familiarity with the problems of trauma and their management. Civilian training programs for first aid and emergency care under the stimulating influence of the Civil Defense Administration, Red Cross, Public Health and similar organizations grow in popularity. Fires, floods and tornadoes have pointed out alarming deficits in organization and skill for effective management of the large numbers of casualties they suddenly create.

Military medicine of necessity specializes in the problems of trauma and preventive medicine. Principles, doctrines and dogma have been evolved, mostly the result of hard earned experience, by conscientious authorities who have served in medical support of a military mission. Study and research continue in this field by both the military and civilian institutions. Many of these principles are applicable to civilian medicine and this is certainly true under conditions of mass disaster.

The military surgeon rapidly learns that he is part of a team. He also learns the value of early emergency care, resuscitation, triage and the necessity of constant evacuation rearward to other members of the team and other facilities. He particularly learns that his judgment may determine life or death to the many injured passing through his assigned station in the chain of evacuation. He knows that the success of his efforts in large measure is dependent upon the judgment of the member of the team preceding or following him in the chain, one whom he may replace another day. To be effective he becomes proficient in his position on the team. Above all he develops a healthy respect for trauma and humility for his inadequacies.

In the event of mass disaster these principles and lessons of military surgery will be applicable. Whenever the number of casualties exceed the available medical means, only through efficient organization and teamwork can the greatest good be accomplished and the maximum lives and limbs be saved.

The immediate problems of mass casualty care, civilian or military, are those of the treatment of all varieties of injuries and the establishment of effective organization. In atomic warfare traumatic injuries and thermal burns will predominate. There will be multiple injuries and combinations of injuries. Of the mechanical injuries, those of the soft tissues, contusions, lacerations, avulsions, penetrating wounds and amputations will be in abundance in all degrees of severity. Considering the appalling work load and the paucity of trained personnel and medical equipment, initially the challenge becomes one of doing the best for the greatest number by who is available with what is available. Priorities must be established; calculated risks and penalties must be assumed.

Military surgery teaches that all war wounds are contaminated and at most should be cleansed, débrided if possible and dressed but never primarily closed. To do so is hazardous

to life and at least, prolongs morbidity. A properly débrided wound will cleanse itself and usually may be closed surgically in four to seven days. Proper débridement involves removal of all debris and dead and devitalized tissue; incising compartmentalizing fascia, exposing all recesses and instituting counterdrainage if needed. The soiled skin bordering wounds of entrance and exit is trimmed away and the wound walls kept separated by fluffed gauze. Packing or use of antiseptics is prohibitive. Probing of wounds serves no useful purpose. A firmly applied bandage will control oozing and extensive wounds are immobilized. Adequate surgical débridement may not be simple and requires experience and facilities.

It is more than reasonable that under conditions of atomic warfare such ideal treatment will not be possible for hours. Simple dressings must suffice with, at most, removal of obvious foreign bodies and perhaps relaxing incision of the skin and fascia to facilitate drainage. Significant bleeding is controlled by pressure dressing or vessel ligation, the tourniquet being reserved for amputations and hemorrhage which is uncontrollable by simpler means. Under the circumstances only simple procedures may be considered initially, for those more extensive will require additional skill and equipment. Were they available, establishing priority for care must continue.

The dictum to "splint all fractures where they lie" must be carried out without reservation. Failure to do so invites further injury and shock. Standard splints will not be available during the critical hours and improvisation using available material will be necessary. A poorly applied splint is of little value. Efficient traction splintage may not be possible. Open fractures should be dressed as other wounds and immobilized. If possible, a joint capsule should be closed, leaving the remaining wound open. Internal metallic fixation will be avoided. Due regard must be given fractures of the neck and spine, for poor handling resulting in paralysis is a felony.

Normally wounds of the scalp and face may be surgically prepared and primarily closed. However, to close inadequately cleansed wounds of this region is unsound. Basically questions of deformity and disfigurement, regardless of location, are problems of another day. Although dural wounds should be closed, head injuries *per se* are low in priority for repair. The required time makes such consideration impossible and military experience has shown the value of reasonable delay and resuscitation.

Extensive wounds of the jaw and cervical region require early tracheotomy in addition to the immobilization of the former and adequate dressing of the latter.

Open chest wounds must be closed regardless of contamination, for failure to do so is fatal. Hemothorax is best managed by repeated aspiration and observation. Continuing intrathoracic hemorrhage requires open thoracotomy which implies facility and appropriate anesthesia. Pneumothorax is easily managed by an anterior intercostal tube and flutter valve. Closed drainage will prove to be impractical particularly if transportation is required. One must constantly keep in mind that all after-care could be by unskilled personnel in inadequate facilities at remote places for an indeterminate number of days.

Abdominal wounds pose a real problem when masses of casualties occur and supporting facilities are lacking. Normally such cases are high in priority for early surgical care. Again such treatment involves available minimal assistance, adequate facilities, anesthesia and surgical skill. There can be little question that the penalty of delayed surgical care will be severe. Nevertheless most abdominal procedures are time-consuming for penetrating abdominal wounds produce multiple injuries, each of which requires specific technics. Furthermore, the survival rate of patients with such injuries during conventional warfare is notoriously poor. Many of these injuries will prove to be hopeless. One may instruct professional personnel generally in the procedures for satisfactory emergency care of many types of injuries but not those to the abdomen unless the personnel have had previous surgical experience. Poor abdominal surgery, if not useless, is deceptive.

Assuming facilities and organization were available, certain principles pertain and should be strictly adhered to: (1) All wounds of the small bowel and stomach must be closed. (2) All wounds of the large bowel must be exteriorized other than through the wound of entry. (3) Rectal wounds should be closed and proximal colostomy performed. (4) The injured spleen must be removed—never repaired. (5) Injuries to the liver and pancreas should be repaired and drained. (6) Injuries to the bladder should be closed with a protective indwelling urethral

catheter or treated as suprapubic cystotomy. (7) The kidney withstands injury well and survives to function. Conservation is the rule. (8) Laceration of the diaphragm must be closed. (9) All injuries to the gastrointestional system require nasogastric drainage.

Eviscerated abdominal contents are better replaced and the abdominal wound closed, with reliance on the development of localized peritonitis which may be subsequently drained.

The injured hand should be cleansed, dressed and splinted in the position of function. All viable tissue must be preserved and amputation of any element deferred. The thumbless hand is practically useless. A partially functioning hand is better than any prosthetic device. Plastic procedures are problems of another day; and whereas normally palmar and solar flaps should be basted to underlying tissues, to do so in untreated contaminated wounds is hazardous.

Whereas in wound surgery intact nerves and important blood vessels should be protected, severed nerves should be left alone and severed blood vessels ligated. Assuming that adequate time, personnel and facilities are available, major arteries could be repaired and limbs saved. This requires skill and time, both of which are uncertain quantities in the early hours of mass disaster.

Scientists inform us that man has succeeded in perfecting a device whose destructive power is capable of annihilating entire communities. History already records the appalling results of two such experiences produced by primitive models. The public press gives recognition to each report of progress being made in harnessing this power for domestic and commercial use. This same press, however, also records the considered opinions of recognized authorities who predict the almost incomprehensible potential of the more recently developed mechanisms were they employed by man to destroy his fellow man. It is small wonder that statesmen are constantly alert to quarantine any international dispute, however small, knowing full well the enveloping nature of war.

If our nation were to be suddenly attacked by an enemy employing nuclear weapons, it is safe to assume that many areas would be struck simultaneously. The resulting physical and human destruction would be incalculable. The dead and injured would be measured in terms of hundreds of thousands. It is the remaining living with which we are concerned, for they will build a new civilization. Although such a grim picture is unpleasant to contemplate, it could be national suicide to avoid the truth and fail to prepare for such an eventuality.

The role of the medical profession in this regard is clear. Of our heritage, we are rightfully proud. However, we may be judged by generations to follow if we have failed in our duty today. Our nation expects our profession to initiate and supervise broad educational programs in first aid and emergency care. This is not an invitation but a moral obligation. Its effectiveness depends upon universal participation by our profession and a working familiarity with the principles and technics for the management of trauma generally. Effective mass casualty care of the magnitude presented implies acceptance of priorities for initial treatment and definite compromises with accepted penalties if there is to be maximal benefit and survival. Our medical curricula, postgraduate training programs, research activities and all professional congregations must emphasize these subjects, or we fail.

Of this we may be sure, if judgment is rendered we cannot plead we were not forewarned.

Problems Encountered in the Emergency Treatment of Irritating Gas Casualties

LEWIS C. MANGES, JR., M.D. AND EUGENE H. CONNER, M.D., *Philadelphia, Pennsylvania*

ON October 28, 1954, at 6 A.M. the Third Battalion of the Philadelphia Fire Department, consisting of fourteen men, responded to a telephone message stating that "ammonia fumes" were escaping in the vicinity of a chemical and oil manufacturer's plant in the north central portion of Philadelphia. Those companies who first answered the alarm believed that they had discovered the source of the fumes and sent for additional help which included a Rescue Squad. A total of forty-six men including their officers were at the scene. The three senior officers, along with the various members of their command, were inspecting two 4,000 gallon aluminum alloy storage tanks found in the backyard of the chemical plant, when one of the tanks exploded. The three inspecting officers were killed and an indefinite number of the Fire Department personnel were exposed to vaporized chemicals contained in the tank. All Fire and Police Rescue Squads in the surrounding area were immediately called. The problem of transportation and deployment was rapidly and efficiently managed. The three officers were taken to a small private hospital located near the scene of the accident and pronounced dead. Each of five general hospitals within 1 mile of the explosion received some of the injured. The patients were promptly examined and either admitted for treatment or transferred to the Philadelphia General Hospital where the city maintains a thirty-bed ward for the care of policemen and firemen. Twenty-two patients were admitted to this ward. The majority were considered not seriously injured and had been brought directly to the hospital from the disaster area or had been examined in one of the nearby hospitals and thought to be transportable without aggravation of their injuries.

We should like to describe briefly the problems encountered in the treatment of these men at the Philadelphia General Hospital during the first twenty-four hours following the explosion. All these men had chemical conjunctivitis of varying intensity. They were seen promptly by an ophthalmologist who instituted treatment consisting of mydriatics and cortisone combined with a wide-spectrum antibiotic ointment. They were also given dark glasses. There was no evidence of shock nor were there any serious burns of the skin. No patient had severe coughing. On physical examination their lungs appeared clear except for occasional rales. These men were placed in two rooms off the main ward, each of which contained six beds with the overflow going into the main ward. The medical resident was in attendance. At this time we were not familiar with the nature of the chemical agents to which these men were exposed. There was no evidence of systemic toxicity.

Within a few hours it became evident that extensive edema of the mucous membranes of the mouth and pharynx was developing in a number of these men. Approximately five hours following exposure, tracheostomy was performed on one of the patients. In the next three hours four additional patients had this operation. The tracheostomies were performed under local anesthesia with the patients in bed. There were no difficulties encountered with the procedure and no complications developed. The conventional 6 and 8 mm. metal tubes were used.

Shortly after the first tracheostomy the assistance of the Anesthesia Department (which is under the direction of Dr. Eugene Conner) was requested. The men had marked dyspnea, orthopnea and substernal burning pain, and showed signs of upper respiratory tract obstruction. Edema of the tracheal mucosa was evident at the time of the tracheostomies and there was a rapid development of symptoms of pulmonary edema two or three hours following the tracheostomies. The edema of the mucosa of the tracheobronchial tree was treated by nebulization of isopropyl arterenol (isuprel®). Aminophyllin was utilized for its broncho-

dilatory properties, but it was thought that bronchospasm was not a significant part of the respiratory problem. Treatment of the pulmonary edema with positive expiratory pressure was instituted. This was accomplished by substituting short (10 to 12 cm.) No. 10 Magill endotracheal tubes for the conventional tracheotomy tubes. These rubber tubes could be adapted easily to the to and fro soda lime canisters of readily available anesthesia apparatus. The positive expiratory pressure (10 to 15 mm. Hg.) was supplied by partially occluding the tail of the rebreathing bag and adjusting the flow of gases.

These men were supplied with 100 per cent oxygen during the first four or five hours of oxygen therapy. Following the development of substernal burning pain, which is one of the signs of oxygen toxicity, an effort was made to switch them to compressed air. This could not be done safely for they became unsaturated. Oxygen-helium mixture (20 and 80 per cent) was tried in order to reduce the work of breathing, but this produced unsaturation of the hemoglobin and was also discontinued.

By the end of twenty-four hours these patients showed increasing difficulty with the expiratory phase of respiration. Areas of atelectasis were continuously developing and vigorous pounding on the chest with aspiration of the trachea following instillation of physiologic saline solution was necessary to keep a patent airway. The entire Anesthesia Department and all available house officers, both residents and interns, were utilized to care for these men. There were two physicians or a physician and a nurse assigned to each patient at all times. Frequent cardiograms, hematocrit surveys and blood pressure determinations were made.

The chemical solvent to which these men were exposed was Dow Chemical Company's "Dow-4-2-1." This is a standardized mixture of three halogenated hydrocarbons: four parts orthodichlor benzene, two parts propylene dichloride and one part ethylene dichloride. The toxicology of each of these chemicals following industrial exposure is known. However, chemical reaction had occurred within the storage tank, probably increasing the pressure and finally causing the explosion. The exact nature of the chemical to which the men were exposed was difficult to determine. The city chemists believed it was probably hydrochloric acid, aluminum chlorate and possibly phosgene. Some of the men undoubtedly inhaled these chemicals and others probably had them blown into the tracheobronchial tree by the force of the explosion. In any event it was the local irritating effect in the tracheobronchial tree that was causing their difficulties.

Two patients died within thirty hours of the explosion. Tachycardia of 140 to 160 per minute developed in one thirty-six year old patient. An electrocardiogram taken about eighteen hours after exposure showed marked irregularities and evidence of intraventricular conduction defect. This was interpreted as being an old myocardial infarct. He died suddenly twenty-nine hours after the explosion. Autopsy showed sterile necrosis of the mucosa of the tracheobronchial tree and obstructive emphysema. This man died as a result of failure of respiratory function.

The second death occurred one hour later in a younger man who showed definite signs of intense respiratory congestion which could not be relieved in spite of our most vigorous efforts. Autopsy showed similar evidence of sterile necrosis of the mucosa of the tracheobronchial tree, and obstructive emphysema. He also died as a result of failure of respiratory function.

There was a total of ten deaths due to this explosion, three of which were directly due to the explosive force. Two patients died in our hospital and four in other hospitals within thirty hours, all as a result of failure of respiratory function. The final death occurred ten days after the accident with the same autopsy findings except that the necrosis was much further advanced.

The problems we encountered were as follows: (1) identification of the chemical, (2) acute chemical conjunctivitis, (3) edema of the upper respiratory tract, (4) edema of the tracheobronchial tree and (5) pulmonary edema.

We recommend that the following procedures be carried out in the emergency treatment of gas casualties: (1) Well organized prompt professional investigation of the chemical or chemicals involved. (2) Prompt treatment of chemical conjunctivitis with mydriatics and cortisone combined with a wide-spectrum antibiotic ointment; dark glasses. All but five of these men recovered promptly and completely from their conjunctivitis. In the remaining five varying degrees of scarring of the cornea

developed; one may require a corneal transplant. (3) Early tracheostomies. (4) Insertion of large caliber tracheostomy tubes that can be adjusted to anesthesia machines. (5) Oxygen therapy. (6) Frequent stimulation of the cough reflex using strenuous means if necessary. (7) Constant care. These patients would best be taken care of in a well organized recovery room.

This is by no means a completed story. Many more problems developed, and we still do not know the final outcome on some of these men.

DISCUSSION OF PAPERS BY DRS. SHAEFFER, AND MANGES AND CONNER

OSCAR PERRY HAMPTON, JR. (St. Louis, Mo.): I want to compliment Colonel Shaeffer on his excellent presentation of his subject in so short a period of time. I would like to point out something I am sure he did not have time to present. In the event of a nuclear explosion in this country, the predominant type of wound will be the penetrating missile wound. Secondary missiles are created by the atomic explosions. That immediately calls to your attention the analogy between the wound surgery of combat and the wound surgery of an atomic explosion.

Many multiple penetrating wounds look innocuous, but when the appropriate excisions are made for access to the devitalized tissues, quite a wound remains.

The problem of mass casualties has come to the attention of the Committee on Trauma of the American College of Surgeons. Dr. Arnold Griswold, the Chairman, has seen fit to have a subcommittee study this problem. It consists of Colonel Shaeffer, Dr. Howard Snyder and this discussor. (I hope Dr. Snyder will mention his recent experience with mass casualties in the metropolis of Winfield, Kansas; it is most informative.)

I want to take this opportunity to advertise some of the activities of the Committee on Trauma, and this subcommittee, so that all of you will do what you can to promote adequate participation in certain activities.

Next year at each of the five sectional meetings of the College of Surgeons to be held in this country there will be a symposium of about three hours on the management of mass casualties. It should be a splendid educational venture, bringing out many of the teachings which Dr. Snyder has touched on briefly and mentioning other points as well. Both military personnel and civilian physicians will participate, and there will be a talk at each of these programs on the important phase of hospital organization for the management of mass casualties. I believe at least one of these programs will warrant your attendance, and we all would appreciate your "talking it up" so that there will be splendid attendance at all of these meetings.

HOWARD ERROL SNYDER (Winfield, Kan.): Dr. Shaeffer certainly has touched upon the most important problem that confronts American medicine today, and I think we are lagging far behind.

More of us need to hear additional talks by this author. Certainly we have to recognize that every doctor of medicine and every medical student must be taught all of the emergency surgical procedures which he may be called upon to perform in the event of emergency or disaster.

Dr. Hampton referred to the metropolis of Winfield, which has a population of about 10,000 people. This town is just 20 miles from Udall, the location of the hurricane. When this disaster occurred, sixty-five casualties were brought to one small hospital in Winfield and thirty to another, with about thirteen doctors to take care of them.

These patients were managed just as were war casualties: with organization, priorities established and wounds left open. Each person underwent surgery. Operations were completed by 4 P.M., and the patients had started coming in shortly after midnight. This proves that emergency measure can be carried out.

There is one thing Colonel Shaeffer and I are not completely in agreement on, probably because we have not discussed it. He, with his contacts in Washington and the knowledge of what may happen with a thermonuclear bomb or an atomic bomb, visualized millions of casualties and just not enough doctors to go around. I think we must remember that disasters will range from the one I just mentioned at Udall to a nuclear explosion; and disasters, just like patients, must be individualized. Most of the disasters we are going to encounter will permit us to establish priorities such as those we are familiar with and practice, namely, that the first priority wounded get immediate shock care, the best care, and laparotomy for abdominal wounds and abdominal hemorrhage. We will take care of those first, postpone care for the less severely injured until later, and take the minor wounds in turn.

You will find in any situation that there will be some doctors who can perform laparotomy and many more who cannot. You should be able to find that all the doctors there can at least assist in minor surgery, the shock ward or in the pre- or postoperative care of these individuals.

R. ARNOLD GRISWOLD (Louisville, Ky.): I would like to express my admiration for Colonel Shaeffer's presentation and repeat what Dr. Hampton said about the importance of this subject at the sectional meetings of the College where we can reach a great many more people. As someone has said,

this information is not just for atomic or nuclear casualties; disasters are likely to happen in any of our own towns any day.

Not too long ago we had an airplane wreck in Louisville. We thought we had a good emergency set-up with adequate training of our police, and so forth. A C-46 with forty-six people aboard crashed on the runway of our airport. All the county and city police ambulances went there. Not one person had a splint applied. All of these well trained first aiders panicked when they saw more than two or three casualties at the same time.

The passengers were taken to the closest hospital to the airport, and at first were scattered throughout the hospital. Not until the arrival of a doctor who had been chief of surgery at an evacuation hospital during the war did these patients begin to get an adequate triage.

Panic in a situation like this is the most important problem we have to deal with. These people were all educated; they knew first aid and had been trained in it, but they all panicked—not only the policemen but also the doctors and interns in the hospital.

Regarding the second paper on chemical poisoning, I should like to point out that this explosion was very similar to the one at Cleveland Clinic in the 1920's when x-ray films caught on fire and nitric oxide gas went through the ventilating system of the clinic. When this gas came in contact with water in the lungs it turned to nitrous acid, producing the same effect the halogenated compound did in Philadelphia.

G. W. N. Eggers (Galveston, Tex.): I had the opportunity of being present at the Texas City disaster. In fact, I was in Texas City forty minutes after it happened and the confusion was great. I was very much impressed by the reaction of the people. They acted as if they had had about 3 gr. of sodium amytal or some other sedative. They were lethargic as a result of this great shock.

This terrific blast which occurred in Texas City killed 563 people in about two or three seconds. To give you an idea of the tremendous force, perhaps you know that a freighter steamship has an anchor weighing close to a ton. The mud hook from the ship that exploded now rests in the American Oil Company yard more than a mile from the ship.

The injured were brought to the hospital. In Galveston, which is just across the bay, a double highway was immediately used. The local civilian defense police department kept this highway open with good organization, allowing the traffic to go back and forth.

About 800 serious cases were admitted to the hospital I was with, and the less serious, an additional 500, were put in buses and taken to the military camp. Without this military help, I do not know what we would have done.

We lacked sufficient supplies. Fortunately we had cots to take care of the people because in the defense program there are accommodations for 350 cots, and the people of the city brought in additional supplies.

I do not want to leave the impression that everything went smoothly, because it did not. The greatest difficulty was, I think, immediately afterward when they started to bring the people to the hospital. None of the fractures had been splinted.

In Texas City all the ambulances were destroyed in the explosion, and the victims arrived in all sorts of conveyances. I do not believe I saw one patient come in with a splint applied. The truth of the matter is that in Texas City they had no splints; they had nothing.

I went to Texas City. We gave a great deal of morphine to help the people who were sitting and lying around, still unattended. The living and the dead caused a confusion which was tremendous. However, within two hours the Army was flying in blood plasma and supplies, and staffing and assisting.

We were fortunate in having at our institution the recently discharged 127th U. S. Hospital Unit. Colonel Moore was in charge of the supervision of the injured patients. We separated those who needed immediate attention. We organized traction, operative and plaster teams. The débrided wounds were left open, except for some we "inherited" that were closed by others.

I do not think it is possible to give you a picture of the confusion that took place. The moment of catastrophe is as Colonel Shaeffer described. Regardless of what you tell the people, the first thing is to try to get them somewhere; and if they do not have conveyances, it is a real problem.

Before we were through we had passed from 1,000 to 1,200 patients through the hospital, which I assure you takes a lot of work and a lot of time. The confusion can never be controlled or abolished, only minimized.

Edgar L. Gilcreest (San Francisco, Calif.): It has been a source of great personal satisfaction to me, as I know it has been to Dr. Carothers, to hear these outstanding papers on the broad subject of trauma. It shows us how far our Association has evolved since it was organized in San Francisco in June, 1938.

Prior to its organization, Dr. Carothers and I reviewed the programs for the past five years of five of the leading surgical associations in America and found that less than 10 per cent of the total number of papers read before these associations were concerned with trauma.

We also learned that most of the medical schools did not teach the subject of trauma as they did other divisions of surgery, yet most of their graduates, when they entered practice, had to treat more patients with trauma than any other group.

I well remember that thirty-five years ago surgeons who treated patients who had been injured in industry were known as industrial surgeons and were looked upon as not being scientific men. In many cases that was true because their remuneration was small. Today this has changed and, in most instances, the industrial injured receive scientific care. I further remember that not until our Association was organized did the American College of Surgeons have a committee on trauma, only a fracture committee. I have always maintained that when a patient has a fracture of a bone, he usually has injury of the soft tissues as well and therefore should not always be classified as simply a fracture case.

We have now in our Association some of the finest surgeons on this continent and their contributions are outstanding. This is eloquent proof that the American Association for the Surgery of Trauma was urgently needed and that it has taken a high place among the scientific associations in this country, I am personally very happy to have played a role in its organization.

Robert H. Kennedy (New York, N. Y.): I have had the feeling for the last two or three years, at least, that the term "mass casualties" turns away many doctors, particularly surgeons, from attending that part of a meeting. They think that they will hear a talk about the atomic bomb, and they do not believe that we are going to be bombed.

That is not the entire point which Colonel Shaeffer is stressing. We have to be ready if bombing occurs, but we must also consider what happens in our own hospitals when ten, twenty or thirty casualties are admitted as a result of ordinary civilian disasters.

I hope that the Sectional Meetings of the American College of Surgeons this next year, where this will be a major subject, will be attended by doctors who will not turn away because they think the subject of mass casualties concerns only bombing. It does not take too many injured persons arriving at any hospital to make a mass casualty problem for that hospital.

Joseph R. Shaeffer (closing): I would like to call your attention to an editorial by Dr. Isidor Ravdin in the October issue of *Surgery*. This editorial quotes a letter received from a young surgeon who, having completed his residency, was assigned to a busy military station type hospital as chief of surgery. The young doctor's comments are very interesting.

In this regard I would like to call your attention to the future of all students choosing medicine as a profession. With the probable expiration of the Doctor Draft Law next year all graduating medical students may anticipate two years of military service because of their deferment from the regular draft at the age of eighteen for advanced education. This will mean that the military Medical Corps will be made up preponderantly of these young men, the balance being career officers. Some may be deferred for some period of residency training.

If this is true it would seem reasonable that a professionally competent regular corps should be maintained to guide the development of these doctors during this two-year period.

I agree with the remarks made by Dr. Kennedy, for the principles outlined in my paper apply equally well to military and civilian disaster. In atomic war many areas could be struck simultaneously and target areas could be industrial centers. For planning purposes, current thinking visualizes seventy major cities with a possible 9,000,000 surviving casualties in need of care. The real problem in any disaster is management in the first forty-eight to sixty hours before facilities have been organized.

Treatment of Burns of Difficult Areas

LT. COL. CURTIS P. ARTZ, M.C. AND CAPT. BRUCE G. MACMILLAN, M.C., *Ft. Sam Houston, Texas*

From the Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas.

THE aims in the local treatment of burns irrespective of the area involved are prevention of infection, early skin coverage, minimization of contracture and preservation of function. In certain areas of the body such as the face, hands and feet, and in deep burns involving bone, it is very difficult to achieve these aims. During the past six years more than 900 hospitalized burned patients have been treated in the Surgical Research Unit at the Brooke Army Hospital. Of these 50 per cent involved the face, 44 per cent involved the hands, and 10 per cent involved exposed bone, feet, joints or perineum. About one-half of these patients were burned as a result of either airplane crashes or gasoline fires. Since patients with extensive burns are evacuated to this hospital from all branches of the Armed Services, it is fully appreciated that the foregoing figures do not represent a true sampling of burns in general. However, this experience has made possible an excellent opportunity to evaluate the treatment of severe burns and particularly of burns involving specific problem areas.

Priority of Certain Areas for Skin Coverage. When several areas of the body are burned, certain segments must be given priority for the first grafting procedures. High priority should be given to achieve early skin coverage of the hands and face first, areas about joints and flexion creases second, and finally the flat surfaces such as the limbs and the trunk. (Fig. 1.) Each patient presents an individual problem according to the areas involved and the availability of skin. However, careful planning of skin coverage will minimize residual contractures and preserve maximum function.

Face. Both second and third degree burns of the face do well when treated by the exposure method.[1] Because of the great vascularity of the face, burns of this area are characterized by massive edema in the early phases. Dressings are difficult to apply properly, are extremely uncomfortable to the patient and always present a hazardous situation in regard to respiratory obstruction and aspiration. In preparing the face for exposure the burned areas and surrounding intact skin are cleansed gently with a bland soap, blisters are opened and all detached epithelium is removed. The surface is rinsed with liberal quantities of normal saline, after which the face is left exposed and no applications of any kind are applied.

By forty-eight to seventy-two hours the exudate of a partial thickness burn dries and forms a hard crust. It serves as a natural occlusive protective dressing. Epithelial regenera-

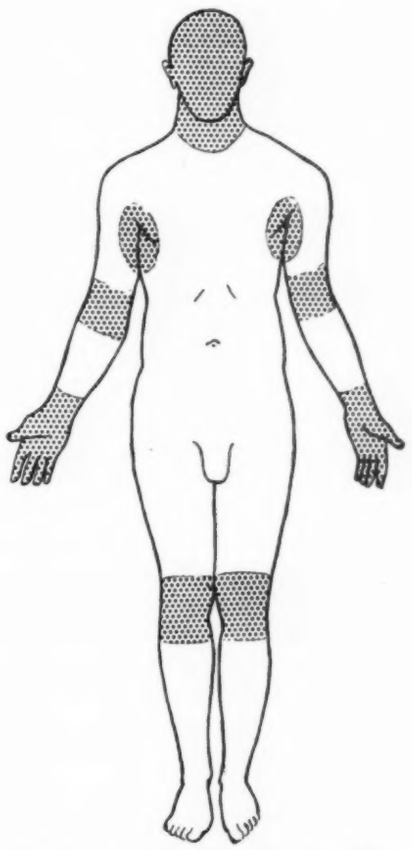

FIG. 1. Certain areas of function should be grafted earlier than other areas. Highest priority should be given to the mottled areas shown in the diagram.

tion proceeds beneath the crust and usually is complete within fourteen to twenty-one days. Full thickness burns present pearly white dead skin which dries and serves as a temporary protective cover. This eschar remains unchanged until liquefaction occurs beneath it as a result of action of leukocytes and bacterial enzymes. Between the eighth and tenth day wet dressings can be applied to prepare the surface for early grafting. Excision of the eschar is rarely indicated.

Indications for Tracheotomy. A tracheotomy is indicated: (1) in respiratory tract burns as evidenced by redness in the posterior pharynx, hoarseness or history of inhalation of hot fumes, and (2) in full thickness burns of the face to prevent sudden respiratory obstruction and to facilitate tracheobronchial toilet. Tracheotomy in burns of the face need not be an emergency procedure. It usually can be carried out under local anesthesia. The tracheotomy facilitates the safe administration of future anesthetics.

Eyelids. Burns of the sclera and cornea are rare. Occasionally these areas are affected in flash burns but the damage is a mild irritation that usually heals within forty-eight hours. However, burns of the lids are quite common. In full thickness burns of the eyelids, contracture occurs which leads to drying of the cornea and ulcer formation. This ulcerated area may become infected, leading to panophthalmitis. The aim in treatment of burns of the lids is to protect the cornea. During the edematous stage infection can be prevented by irrigation and the use of an antibiotic ophthalmic ointment.

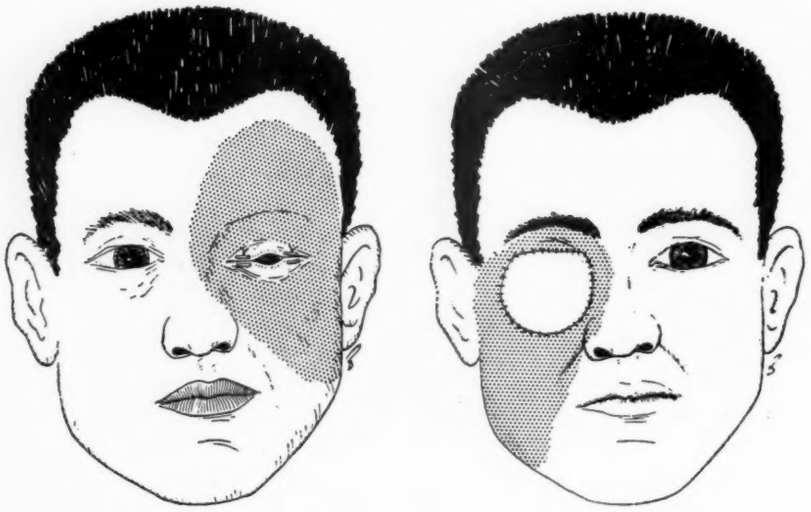

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FIG. 2. Contracture of eyelids is influenced by contracture of the surrounding skin. When the forehead and cheek, along with both eyelids, are involved in third degree burns, a tongue and groove type tarsorrhaphy is indicated at the time of grafting. This will prevent ectropion and drying of the cornea.

FIG. 3. When only one area adjacent to the eyelids is burned, as shown in the diagram, the lid may be overcorrected at the time of grafting. By overcorrection, subsequent ectropion is prevented.

As soon as the eschar is removed from the lids, a graft should be applied. The grafting should be carried out in such a way as to prevent later contracture of the lids or ectropion formation. If the burn involves areas surrounding the upper and lower eyelids (Fig. 2), a tongue and groove type tarsorrhaphy should be carried out at the time of grafting.[2] Tarsorrhaphy is essential or contracture of the surrounding skin will lead to ectropion and its complications. Simple blepharorrhaphy will not prevent ectropion formation because it will pull apart.

The amount of contracture of the lids depends upon the amount of surrounding skin involvement. If the skin adjacent to the lower lid is burned but skin adjacent to the upper lid is normal, tarsorrhaphy is unnecessary. In such

instances the lower eyelid is overcorrected at the time of grafting to allow for some contracture. (Fig. 3.) A better take of such grafts is seen when a stent is applied and the lids are sutured together for temporary immobilization.

Burns of the Ears. Full thickness dermal injury to the external ear results in destruction of the cartilage and infection. As infection progresses, more cartilage is liquefied and an abscess of the auricle results. The aim in such burns is to minimize infection and further destruction of the cartilage. Although not entirely satisfactory, the best method of treatment seems to be the early and wide incision of the rim of the ear. By making a fish-mouth type of incision beginning at the helix and carrying it down over the edge of the auricle, the devitalized cartilage may be exposed and removed. Free drainage can be accomplished by separating the anterior and posterior layers of the auricle with loose packing and the application of continuous warm, wet compresses. The warm compresses also alleviate pain. As soon as the infection has subsided, the ear heals by secondary intention. Almost all full thickness burns of the ear require subsequent reconstructive procedures.

Burns of the Hands. Highest priority should be given to treatment of burns of the hands. In no other area is it so important to obtain early skin coverage in order to achieve a good functional result. Superficial and deep second degree burns of the hands do well when treated by exposure. The patient is encouraged to maintain his hand in a position of function until the crust is formed. As soon as the crust begins to disappear with formation of new epithelium beneath it, motion should be encouraged. Supervised active physiotherapy and whirlpool baths aid in the return of function.

Full thickness burns of the hands are best treated by massive occlusive dressings. Dressings are preferred to exposure treatment because: (1) position of function is better maintained, (2) demarcation between second and third degree areas is evident earlier, and (3) separation of eschar is more rapid. The dressing should be applied in such a way that there is moderate dorsiflexion of the wrist, moderate flexion of the fingers and the thumb is held in a position of abduction and opposition. Special attention should be directed to maintain the width of the web space between the index finger and the thumb.

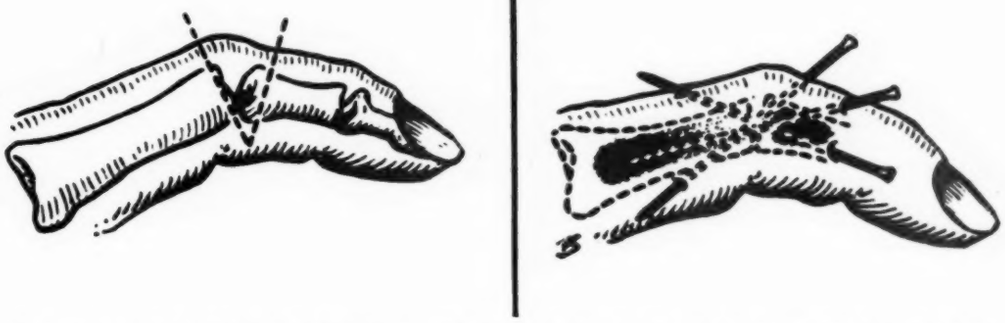

FIG. 4. In very deep burns of the fingers, when pyarthrosis of the proximal interphalangeal joints and destruction of the extensor tendon are present, the joints should be fused at the time of grafting. Fusion in a position of function, by the use of wires after excision of the articular cartilage, prevents flexion contractures.

In some localized full thickness burns of the hand, excision of the dead tissue may be performed by the third or fourth postburn day. This excision can be carried out as soon as the area of full thickness injury is demarcated. Early grafting following excision permits early motion and more rapid return of function.

In deeper burns of the hand when extensor tendons or joints are involved, additional procedures are indicated to preserve maximum function. Non-viable tendons are débrided so as to hasten skin coverage. When pyarthrosis of the proximal interphalangeal joints with loss of the extensor tendon occurs, these joints must be fused in a position of function to prevent severe flexion contractures. This procedure should be carried out at the time of grafting. The articular cartilage is removed and approximately 30 de-

grees of flexion maintained by the insertion of Kirschner wires. (Fig. 4.) Fusion becomes solid in approximately four to six weeks, at which time the wires can be removed.

Third degree burns of the palm of the hand and the plantar surface of the feet are not common. When they do occur, the area should be grafted initially with split thickness skin. This is usually sufficient. Occasionally, however, thicker skin must be applied at a later date.

SUMMARY

In the management of more than 900 hospitalized burned patients certain technics have been evolved for the treatment of specific problem areas. Priority for skin coverage must be given to the face and hands. Exposure is indicated in all burns of the face and in second degree burns of the hands. Dressings are preferred for third degree burns of the hands. Ectropion and its complications can be prevented by tarsorrhaphy. Full thickness burns of the ears are treated by a fish-mouth incision, débridement and wet dressings. Tracheotomy is suggested for respiratory tract burns and all full thickness burns of the face. Kirschner wire immobilization and fusion of the involved proximal interphalangeal joints minimize flexion contractures. Early application of thick, split thickness skin to the palms of the hands and soles of the feet is usually sufficient.

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The Effect of Stress, the Adrenal and the Pituitary on Healing

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The success or failure of healing is of fundamental importance to the surgeon, and often is a measure of his triumphs and disappointments. The healing wound is an intense metabolic focus sensitive to many factors and regulatory mechanisms. We thought a brief review of animal data illustrating the effect of stress, the adrenal and the pituitary on the healing process would be of interest.

Workers in our own and other laboratories have offered substantial evidence that adrenal cortical steroids exert an inhibitory influence on the healing wound. Similar inhibition is evident following administration of ACTH. This pituitary hormone exerts its effect by virtue of its ability to stimulate production of adrenal cortical steroids. The usual physiologic reaction to stress entails increased production of ACTH and adrenal cortical steroids. Each day the surgeon encounters the patient who

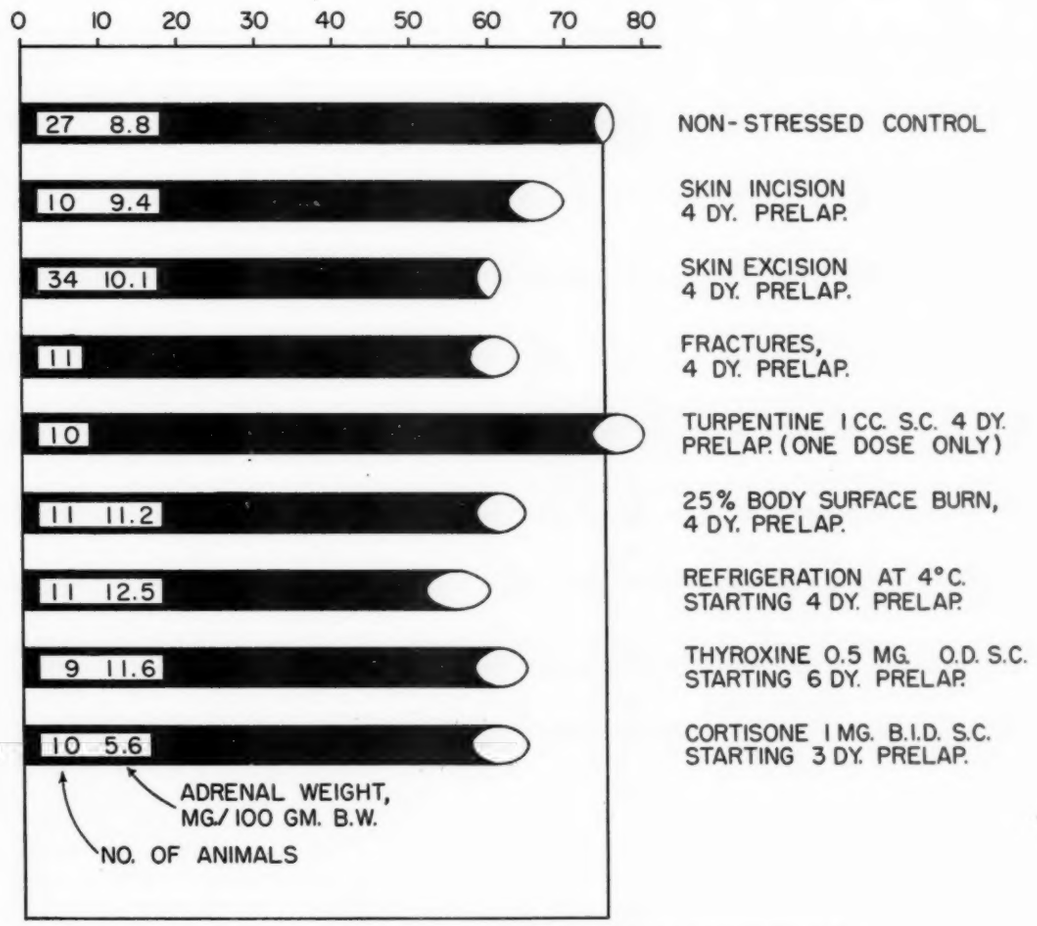

Fig. 1. Mean bursting pressure of fifth-day laparotomy wounds.

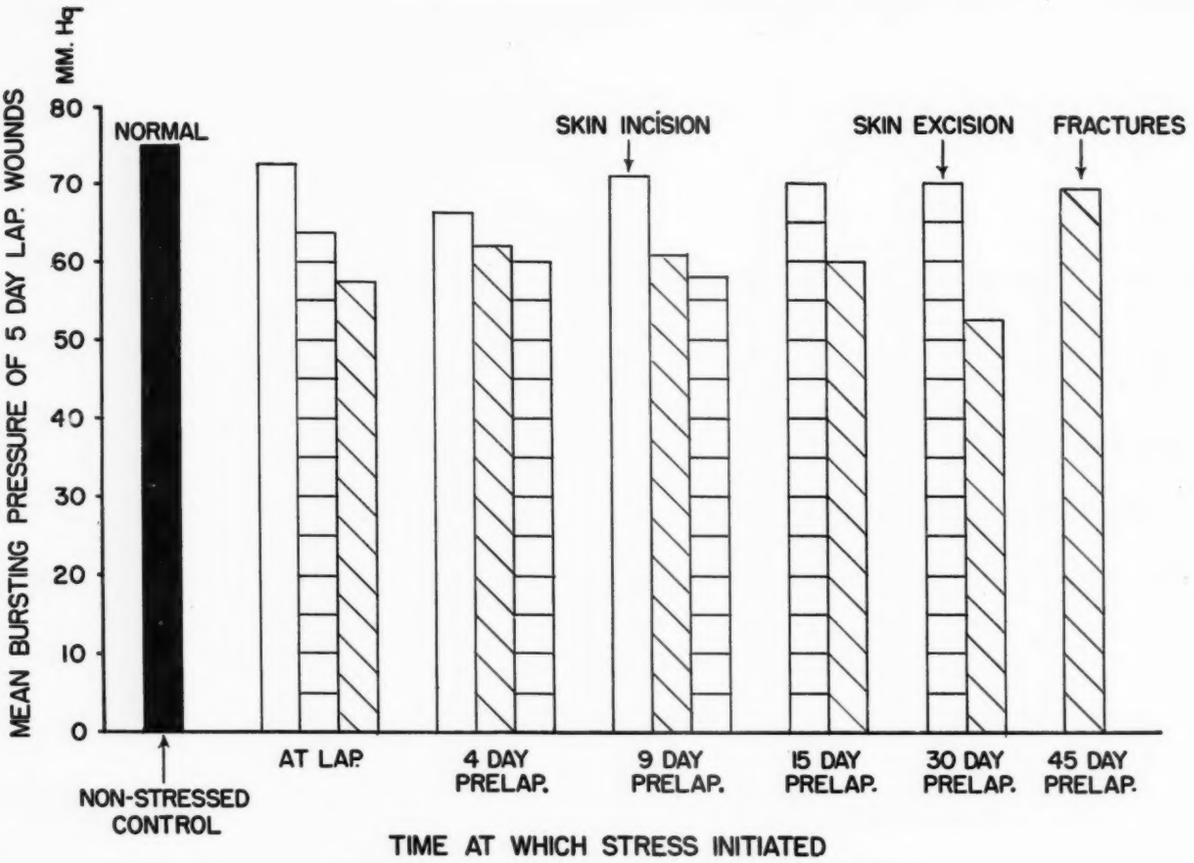

FIG. 2. Duration of stress-induced inhibition of healing.

exhibits this physiologic reaction to stress. Each day the surgeon intensifies the stress phenomenon by the very procedures that are his therapeutic tools. The following theoretic questions arise: (1) Can the normal adrenal gland stimulated by stress produce a sufficient amount of cortical steroids to inhibit healing? (2) If inhibition of wound healing is possible under such conditions, several other queries will be in order. Is the inhibition proportional to the intensity of the stress, how is it mediated and how long does it persist? We shall attempt to answer these questions with illustrative animal data.

For the purposes of brevity we shall not detail the experimental methods. They have been used in our laboratory for fourteen years and have been reported elsewhere. We believe they offer a reproducible and reliable method of measuring the tensile strength of a wound. All data to be discussed are significant and have been submitted to analysis.

Figure 1 charts the mean bursting pressure or tensile strength of the fifth day laparotomy test wound in normal and stressed rats. The number of animals in each group and the mean adrenal weight are listed. Minor stress, such as simple skin incision or the injection of 1 cc. of turpentine subcutaneously, produced no significant depression of healing of the fifth day test wound. More severe stress such as fracture of all four extremities, excision of a patch of skin 6 by 3 cm. or 25 per cent body surface burn produced depression of healing and an increase in adrenal weight. Stress produced by means not primarily associated with blood and plasma loss was found to be capable of producing inhibition of healing associated with adrenal hypertrophy. This is illustrated by data on animals receiving 0.5 mg. of thyroxine subcutaneously for six days prior to making a test wound and for the five days prior to sacrifice of the animal for testing. Refrigeration of animals at 4°C. continuously for nine days produced marked depression of healing and adrenal hypertrophy.

In summary therefore, these experiments indicate that various varieties of stress asso-

ciated with adrenal hypertrophy result in inhibition of healing. Generally the more profound the stress, the more profound the depression of healing and the more marked the adrenal hypertrophy.

In Figure 2 we compare the length of time

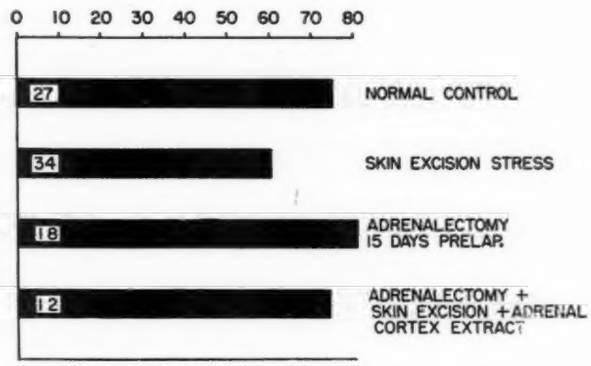

FIG: 3. Stress and adrenalectomy.

that depression of healing is associated with mild, moderate and severe stress. Simple skin incision, excision of a patch of skin 6 by 3 cm. and fracture of bones in all four extremities have been chosen as examples of these varieties of stress. The effect of skin incision is not significant. The recovery from the inhibitory influences of moderate, or skin excision, stress, occurs in fifteen days; the recovery from multiple fractures, or severe stress, requires forty-five days.

Data in Figure 3 compare healing of the fifth day test wound in normal animals, stressed animals, adrenalectomized animals and adrenalectomized stressed animals; they also demonstrate the absence of the stress inhibition of healing in the adrenalectomized animal. The adrenal gland is, therefore, an essential link in the chain of the depression of healing secondary to stress. In order to complete this experiment it was necessary for survival of the adrenalectomized stressed animal to support it with a small fixed dose of adrenal cortical extract.

In an attempt to delineate further the effect of stress-induced inhibition of healing and its relation to adrenal function, the obvious experiment would be one utilizing the stressed animal unable to undergo adrenal stimulation. To this end we studied healing in the hypophysectomized animal. On the basis that the adrenalectomized stressed animal does not demonstrate inhibition of healing, one would, in similar experiments, expect the same findings in the hypophysectomized animal because of the inability to produce ACTH.

The data in Figure 4 indicate that the very opposite occurs, and the test wound in the hypophysectomized animal is markedly in-

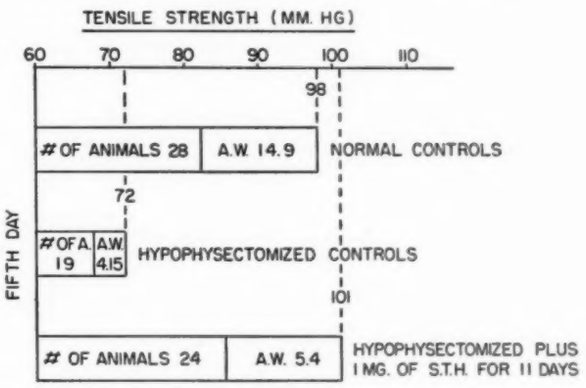

FIG. 4. Effect of hypophysectomy and somatotrophin on healing.

hibited as compared to the normal controls of the same age and strain. These data would indicate that the pituitary produces not only ACTH which can inhibit healing through the adrenal, but also some other substance that is supportive to the healing process. The bar graph illustrates a normal tensile strength for the test wounds of hypophysectomized animals that received 1 mg. of somatotrophin or growth hormone (Armour) for eleven days. It would appear that the growth hormone is the healing support hormone produced by the pituitary.

We realize the danger of blind transference of animal data to human problems. However, certain speculations regarding healing are suggested by the foregoing animal experiments:

1. The more severe the stress, the greater our wound-healing problems should become.

2. In the face of severe stress we should examine our attitude regarding the optimum time of staging operations.

3. The concept that the mobilization of protein fragments as a result of stress might be advantageous to the healing wound is not supported by our data, which indicate that the adrenalectomized animal which presumably does not mobilize protein as a result of stress heals as well or better than his non-adrenalectomized brother.

4. Growth hormone has a supportive function in the processes of healing.

DISCUSSION OF PAPERS BY DRS. ARTZ, PILLSBURY AND MACMILLAN, AND LOCALIO, CHASSIN AND MACKAY

LESTER BREIDENBACH (New York, N. Y.): I think this work is important in that everything the surgeon does, of course, has to do with healing. It has occurred to me that in many individuals who are severely injured a great deal of the corticoid substance was thrown out by the individuals indicating very severe stress.

Since severe stress is markedly inhibitory, by the hormone action, it certainly delays healing. The more severe the stress, the longer the time, the greater the persistence of the stress factor of the hormone secretion.

This, I believe, in a great many instances accounts for non-union in so many cases of fractures because in the individual with severe stress, whether it be that particular fracture or multiple injuries which are often associated with it, there is a tremendous amount of inhibition. This inhibition alone, despite the fact that the fracture is properly handled, may cause it to go on to non-union.

I believe one other important thing about this work is the growth hormone, whatever that may be at the present time; although it has been crystallized, it may be a very important factor in this whole keystone. From this growth hormone, which probably causes inhibitory powers, we may be able to find an antigrowth hormone also secreted by the pituitary, and in this way give some therapeutic value to the surgeon's work.

FREDERICK LEET REICHERT (San Francisco, Calif.): The last paper dealt with normal animals. We have among our patients who come to operation those who are depleted and those with depleted pituitary.

Dr. Localio and co-workers mentioned that recovery from multiple fractures required forty-five days. Were the animals stressed again? That would be a good problem because then the authors probably depleted something in those animals. We certainly have found that with adrenal cortex we can help the human who we know has a depleted adrenal gland, and very often we can relieve his pain through surgery.

Nobody mentioned pain, which is a great stress. Burned patients certainly have pain.

ARAN S. JOHNSON (Detroit, Mich.): If I may be given the permission to discuss the paper by Dr. Localio and his co-workers, we had the opportunity in 1952 of doing some work with cortisone in decreasing the size of myocardial infarcts and the mortality rate. After operating on over 200 dogs, we noticed absolutely no interference with wound healing. I can see no relationship as far as stress is concerned in the production of corticoids and its interference in wound healing.

In our experiments we found that cortisone and ACTH physiologically did not interfere with wound healing in over 200 experimental dogs.

I very seriously question Dr. Localio's premise as far as his paper is concerned.

We have also used Armour's growth hormone alone and in combination with cortisone and ACTH, which has increased the healing period in our experiments in dogs.

J. W. BLUNT (Cleveland, Ohio): The first thing we must do is realize some of the problems which arise when we talk about the use of hormonal materials in wound healing. We speak entirely of measurements of levels of the hormones and the levels in urine, yet we still have no understanding about how the hormones act. We do not know the mechanisms of how cortisone works on a wound, how it inhibits healing or how it may promote it.

It is interesting to make observations on these other findings as far as burns and wound healing are concerned. However, before evaluating anything which is reported from animal work, I would like to remind you of one very important factor—a dog is a notoriously poor animal on which to use any steroids.

In order to get an adrenal effect from cortisone or from ACTH, one has to use massive doses. By that I mean 10 mg. per kg. of weight, whereas the human dose ranges somewhere around less than ½ mg. per kg. to get an effective dose.

The rat is that much higher than a mouse that we must use the equivalent of 60 mg. per kg. in order to get an effect.

These data must be evaluated; but when one is considering the use of animal experimentation and interpreting it in humans, I just wanted to bring that point up so we would draw no false conclusions.

S. ARTHUR LOCALIO (closing): We have not done the experiment that Dr. Reichert suggested. He did touch on a very important point—the problem of the depleted patient in wound healing. This is infinitely more important to adequate wound healing than some of the aberrations of hormonal balance.

Our desire in bringing this paper to you is to try to indicate that there is more to wound healing than just putting a dressing on and praying that it will heal.

The wound is an intense metabolic focus that is sensitive to many factors. It is sensitive to protein depletion, and in our animal experiments we tried to control this depletion factor as best we could.

In an attempt to control the pituitary experiment, done with growth hormone, we did paired feeding experiments so that both the hypophysectomized and the adrenalectomized animals that received growth hormone had the same caloric and protein intake.

Dr. Blunt was kind enough to answer Dr. Johnson's question for me.

Human Bites, a Series Treated with Antibiotics*

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SINCE the classic paper of Mason and Koch[1] in 1930 on human bites there have been reports of approximately 1,000 cases of bites in humans. These subsequent papers have served to establish the astuteness of the observations of these early investigators and prove the value of their plan of treatment. The advent of antibiotic therapy, however, has given us a powerful supplement to the classic treatment of these lesions and has altered the prognosis to a considerable degree. Because very little material has been presented from the antibiotic era, a report of a new series seems appropriate.

Prior to 1930 the literature on human bite wounds consisted of scattered reports covering approximately thirty-five cases. In 1930 Mason and Koch[1] reviewed thirteen cases which they personally treated, and correlated their observations with experimental studies on cadavers after the method of Kanavel. They established the special significance of the injury caused by striking the closed fist against human teeth. The more serious nature of this injury has been repeatedly observed and is now widely appreciated. They also outlined a plan of treatment for early wounds consisting of soap and water cleansing, conservative débridement and leaving the wound open. Continuous moist dressings with the hand splinted in the position of function were also suggested.

After the singular nature of the human bite wound had been set forth in the Mason-Koch paper, numerous reports began to appear from all parts of the country. In Table 1 are recorded the larger series that have appeared through the years. In most of these papers the principles of Mason and Koch are reiterated. There are, however, two notable exceptions. Bates[2] recommended electrocautery of all early wounds, and Lowry[4] recommended cautery with silver nitrate. Neither of these methods seems to have attracted any significant number of followers, and both are now generally put aside in favor of the earlier routine of soap and water cleansing and débridement.

The use of sulfa drugs and antibiotics was beginning to appear in 1948 when Boyce[14] reported his second series from New Orleans. Besides reporting ninety-three new cases, he reviewed the literature to that point and summed up the significant facts. The bacteriology had become better understood. The most common organisms were Staphylococcus aureus and albus, non-hemolytic streptococcus viridans, micro-aerophilic streptococcus, Bacil-

TABLE 1
HUMAN BITE SERIES IN THE LITERATURE (1,030 CASES)

Author	Year Reported	Source of Series	No. of Cases
Mason and Koch[1]	1930	Chicago, Ill.	13
Bates[2]	1931	Philadelphia, Pa.	100
Welch[3]	1936	Massachusetts General Hospital	18
Lowry[4]	1936	Beekman-Downtown Hospital, N. Y.	122
Maier[5]	1937	Bellvue Hospital, N. Y.	27
McMaster[6]	1939	Los Angeles, Calif.	68
Cohn[7]	1940	San Francisco Hospital	35
Fritzell[8]	1940	Minnesota General Hospital	20
Boland[9]	1941	Grady Hospital, Atlanta	60
Speirs[10]	1941	Detroit Receiving Hospital	114
Miller and Winfield[11]	1942	Detroit Receiving Hospital	115
Boyce[12]	1942	Charity Hospital, New Orleans	90
Hudson[13]	1944	Long Island, N. Y.	14
Boyce[14]	1948	Charity Hospital, New Orleans	93
Grimes and Manges[15]	1949	Philadelphia General Hospital	68
Crikelair and Bates[16]	1950	Detroit Receiving Hospital	46
Levin and Longacre[17]	1951	Charity Hospital, New Orleans	27

* This paper was awarded a prize as the best essay representing original research in the field of trauma.

TABLE II
HUMAN BITES

Case No	Age (yr.), Race and Sex	Late (Days)	Days in Hospital	Position of Bite	Blow Injury	Reason for Admission	Suture	Amputation	Fracture	Osteomyelitis	X-ray (x) Culture (c)	Pen.cillin	Streptomycin	Aureomycin	Terramycin	Achromycin	Chloromycetin
Patients Admitted to Hospital during 1953 and 1954																	
1.	59, N., M	..	6	Rt. hand, arm, thigh	..	Inflammation	..	.....	...	..	x	x	x	..	..	..	..
2.	55, W., M.	..	7	Rt. finger	..	Alcoholism	..	.....	...	..	...	..	..	..	..	..	..
3.	63, N., F.	..	1	Lt. thumb	..	Head injury	..	.....	...	..	...	x	x	..	..	..	..
4.	42, W., M.	..	5	Lt. thumb	..	Inflammation	..	.....	...	..	...	x	x	..	..	..	..
5.	34, N., F.	..	5	Face	..	Débridement	x	.....	...	..	...	x	x	..	..	..	..
6.	33, W., M.	..	5	Rt. thumb	..	Débridement	..	.....	...	..	...	x	x	..	..	..	..
7.	30, N , F.	..	1	Lt. finger	..	Débridement	..	By bite	...	..	x	x	..	x	..	..	..
8.	17, W., M.	..	4	Lt. hand	x	Inflammation	..	.....	...	..	...	x	x	x	..	..	..
9.	29, N., F.	..	8	Lt. finger, thumb	..	Inflammation	..	.....	...	..	...	x	x	..	..	..	..
10.	17, W., M.	..	1	Rt. hand	x	Diabetes	..	.....	...	..	...	x	x	..	..	..	..
11.	42, W., M.	..	1	Rt. hand	..	Gunshot wound, foot	x	.....	...	..	...	x	x	..	..	..	..
12.	37, W., M.	..	5	Lt. hand	x	Débridement	..	.....	Fx.	..	x	x	x	..	..	..	..
13.	28, N., F.	..	7	Rt. thumb	..	Débridement	..	.....	Fx.	..	x	x	x	..	x	..	..
14.	29, N , F.	..	1	Forearm	..	Inflammation	..	.....	...	..	...	x	x	..	..	..	..
15.	55, W., M.	18	31	Lt. finger	..	Abscess, incision and drainage	..	Surgery	Fx.	x	c, x	x	..	..	x	..	..
16.	43, N., F.	30	6	Lt. finger	..	Abscess, incision and drainage	..	.....	Fx.	x	c, x	x	x	..	..	..	..
17.	29, W., M.	?	?	Lt. finger	..	Abscess, incision and drainage	..	.....	...	..	c, x	..	..	..	..	..	..
18.	30, N., M.	6	8	Lt. forearm, thumb	..	Cellulitis	..	.....	...	..	c, x	x	x	x	..	..	..
19.	19, W., M.	10	3	Lt. hand	x	Cellulitis	..	.....	...	..	x	x	x	x	..	..	x
20.	39, N., F.	10	26	Rt. hand	..	Cellulitis	..	.....	...	..	c, x	x	..	..	x	..	..
21.	34, N., M.	9	7	Lt. shoulder, arm, finger	..	Cellulitis	..	.....	...	..	...	x	x	x	..	..	..
22.	49, W., M.	9	26	Rt. finger	..	Cellulitis	..	.....	...	x	c, x	x	x	..	..	..	..
23.	23, N., M.	10	5	Rt. hand	x	Abscess, incision and drainage	..	.....	...	..	c, x	x	x	..	..	..	..
24.	40, N., M.	4	5	Forearm	..	Cellulitis	..	.....	...	..	c, x	x	x	..	..	..	..
25.	40, N., M.	21	1	Lt. finger	..	Abscess, incision and drainage	..	.....	...	x	x	x	x	x	..	..	..
26.	30, N., M.	4	3	Rt. hand	..	Abscess, incision and drainage	..	.....	...	..	...	x	..	..	..	x	..
27.	52, N., M.	9	38	Rt. hand	x	Cellulitis	..	.....	...	..	...	x	x	..	..	..	..

TABLE II (*Continued*)

Case No.	Age (yr.), Race and Sex	Late (Days)	Days in Hospital	Position of Bite	Blow Injury	Reason for Admission	Suture	Amputation	Fracture	Osteomyelitis	X-ray (x) Culture (c)	Penicillin	Streptomycin	Aureomycin	Terramycin	Achromycin	Chloromycetin
Patients Seen by Author in 1955																	
28.	38, N, M.	36	2	Rt. hand	x	Cellulitis	..	.....	...	..	x	x	x	..	..	..	..
29.	19, N., F.	2	..	Rt. finger	..	.....	..	.....	...	..	c	x	x	..	..	..	..
30.	29, N., F.	..	1	Lt. finger	..	Drug reaction	..	.....	...	..	...	x	x	..	..	x	..
31.	25, W., M.	..	8	Lt. thumb	..	Urticaria	..	.....	...	..	...	x	..	..	..	..	..
32.	14, N., F.	..	..	Both cheeks	..	.....	..	.....	...	..	...	x	x	..	..	..	..
33.	19, N., M.	..	..	Lt. finger	..	.....	..	.....	...	..	...	x	..	..	..	..	..
34.	47, N., F.	..	..	Rt. finger	..	.....	..	By bite	...	..	...	x	x	..	..	..	..
35.	38, W., M.	5	2	Rt. finger	..	Abscess, incision and drainage	..	.....	...	..	c	x	..	..	..	..	..
36.	41, N., M.	3	..	Lt. hand	x	.....	..	.....	Fx.	..	c, x	x	x	..	..	..	..
37.	22, N., M.	..	..	Ear	..	.....	x	.....	...	..	...	x	x	..	..	..	..
38.	27, N., F.	..	..	Arm, neck	..	.....	..	.....	...	..	...	x	..	..	..	..	..
39.	25, N., M.	..	..	Rt. hand	..	.....	..	.....	...	..	...	x	x	..	..	..	..
40.	29, W., M.	3	..	Rt. hand	x	.....	..	.....	...	..	...	x	x	..	..	..	..
41.	21, N., F.	3	..	Lt. hand	..	.....	..	.....	...	..	...	x	..	..	..	..	..
42.	33, W., F.	2	..	Rt. finger	..	.....	..	.....	...	..	...	x	..	..	..	..	..
43.	31, N., M.	..	..	Lt. hand, arm	..	.....	..	.....	...	..	...	x	x	..	..	..	..
44.	37, N., M.	3	..	Lt. finger	..	.....	..	.....	...	..	...	x	x	..	..	..	..

lus proteus and Bacillus subtilis. The emphasis on the fusiform bacillus and spirilla had waned, but when present they were recognized as suggesting a more serious prognosis. In Boyce's second series sixty-six of the ninety-three patients received penicillin or sulfa drugs or both. The series showed considerably better results than the first group, but the patients were seen earlier on the average and were usually admitted for forty-eight hours to the hospital. The antibiotics could not be considered the cause for improvement, therefore, although it was thought that they definitely contributed.

Grimes and Manges[15] reviewed sixty-eight cases in three groups, pre-sulfa, sulfa and penicillin. Although the numbers were too small for statistical evaluation, there appeared to be a definite improvement when penicillin was incorporated in the regimen. In discussion of this paper, Siler[18] stated that he had been using chloromycetin® and believed that it was even more effective than penicillin. Posch[21] has also had some experience with chloromycetin and has the impression that it is decidedly efficacious.

Levin and Longacre[17] reported twenty-three cases in which treatment used was penicillin, sulfa and streptomycin singly or in combination. They also reported in detail four cases in which intramuscular bacitracin alone was used and found to be effective. Here again, the number is too small to gain more than an impression.

Gardner[19] reported a single case which has the only report of culture with *in vitro*–tested sensitivities found in the literature. He found the three organisms as follows: (1) Friedländer's bacillus—sensitive to chloromycetin, streptomycin, aureomycin, and insensitive to penicillin; (2) Neisseria catarrhalis—sensitive to aureomycin only; (3) Staphylococcus saprophyticus—sensitive to aureomycin, chloro-

mycetin, streptomycin, and insensitive to penicillin. This case was reported largely because of the unusual flora, and therefore gives no guide as to the most probably effective antibiotic for the typical case.

MATERIAL

The present series consists of two main groups of patients. The first is composed of all the admissions with bite wounds to the Detroit Receiving Hospital in the years 1953 and 1954. In this group there are six animal bites, whereas the remainder are human. The human bite cases are summarized in Table II under Case numbers 1 to 27. There is a tendency in the literature to classify bite lesions as immediate, delayed or late. We will refer to cases as immediate if the patient was seen on the day of admission, or late if there were one or more days' delay. In each case the delay is recorded in Table II.

The second group is made up of human bites seen by the author in the clinic or emergency room of the Detroit Receiving Hospital during the first four months of 1955. This does not represent the total number of cases seen in the hospital, but only those which came to the attention of the author. Four of these were admitted to the hospital wards. The 1955 group is summarized in Table II, Cases 28 to 44.

As in many series before, follow-up was incomplete in most cases. The particular social strata in which these lesions occur makes location of patients for late follow-up difficult. When patients were admitted, the wounds were usually followed either in the hospital or clinic to what appeared to be complete healing. Only one patient (Case 27) was readmitted because of inadequate progress as an outpatient. Long-term check was available on one patient. (Case 16).

The patients seen entirely on an outpatient basis in general were never seen with completely healed wounds, but from progress noted had probably failed to return for the last check because the wound had healed. None of these could be located to verify an adequate result.

COMMENTS

Among the bite wounds in hospitalized patients from 1953 to 1954 were twenty-seven human bites and six animal bites. Only one patient of the six with animal bites was admitted because of infection. (Table III.) This is in sharp contrast to the cases of human bites, in which eighteen of twenty-seven patients were admitted for degrees of infection graded from early inflammation to frank abscess. The number of animal bites seen in the emergency room greatly exceeds the human bites. With the reverse true of the admissions, the well known more virulent nature of the human bite is readily verified.

TABLE III

Case No.	Age (yr.), Race and Sex	Animal	Position of Bite	Reason for Admission	Days in Hospital
1.	5, N., F.	Dog	Cheek	Question of rabies	4
2.	5 mo., N., F.	Rat	Hand	Bronchopneumonia	12
3.	9, W., M.	Monkey	Forearm	Plastic repair	1
4.	6, W., M.	Rat	Nose	Inflammation	4
5.	46, N., F.	Dog		Schizophrenia	5
6.	27, W., M.	Dog	Ear	Alcoholism	1

The lesion acquired by striking the knuckles against human teeth, which we have chosen to designate as a "blow" injury, has been noted in column 6 of Table II whenever it occurred. We saw only nine cases in forty-four, a smaller number than was reported in earlier papers. Since this wound is related to fighting, it would be expected to be most commonly on the right hand. Interestingly, in our series involvement of the right hand exceeded that of the left by only five to four. All blow wounds were in men. When all types of lesions were considered, the left hand actually exceeded the right by twenty to eighteen. Only six of the forty-four lesions did not involve the hand. No particular significance seems to be attached to this except that human bites largely involve hands rather than other parts of the body. Men exceeded women twenty-nine to fifteen, and Negro patients exceeded white patients by thirty to fourteen.

For purposes of showing morbidity increase with delay we have established two groups, namely, immediate and late as described heretofore. There seems to be considerable variation in reaction since many wounds two or three days old will show only minimal inflammation; at the other extreme (Case 38, Table II), one patient seen three hours after the injury had frank cellulitis of her arm.

Arbitrary groups are composed with this degree of variation in mind. All four cases of osteomyelitis were in the late group. In two of the three cases of fracture associated with bite in the late group osteomyelitis developed, whereas osteomyelitis did not develop in either of the fractures in the group of immediate care. Days in the hospital also indicate more morbidity in the late group: late: average hospitalization eleven days (range one to thirty-one days); immediate: average hospitalization four days (range one to eight days).

To date there are no reports of tetanus in human bite wounds. Both Lowry[4] and Boyce[14] attach such significance to this remarkable situation that they suggest tetanus antitoxin not be given in these wounds. We have had no cases of tetanus in our series.

In three cases in this series sutures were applied. One patient (Case 11) had his bite wound sutured during débridement of a gunshot wound. The true nature of his bite wound was not appreciated until he was returned to the ward. When the diagnosis was made (about two hours after suturing), the sutures were removed and continuous wet dressings were applied. The patient left the hospital against advice after one day and nothing further is known of him. The other two wounds which were sutured (Cases 5 and 37) involved the face and ear, and the nature of the wound was known in each case.

In Case 5 débridement and irrigation of an infraorbital laceration were done, followed by closure of the wound with sutures. On the second postoperative day the entire cheek was swollen and hot, and the sutures were removed. On the fourth postoperative day the inflammation was localized to the area of the wound. The patient was discharged on the fifth postoperative day and was seen in the clinic with a clean, well healed wound. The other patient (Case 37) suffered a bite of his right ear with exposure of the cartilage and some loss of tissue. His ear was cleansed and sutured in the emergency room and he came daily for penicillin therapy. At the time the sutures were removed, the wound was clean and well healed. The relatively satisfactory course in each of these two cases suggests that there is a basic difference in the course of bite wounds of the hand and bite wounds elsewhere. This coincides with the opinion held by Crikelair and Bates,[16] and is in line with their observations.

In early cases material for culture is usually not obtainable. In our series material was sent for culture in eleven cases. These are summarized in Table IV. The cases have the same number designation as in Table II. In three cases there was no growth. In three more, no sensitivities were tested. In the remaining five, sensitivities were tested *in vitro*. Penicillin, aureomycin and chloromycetin have a rather satisfactory record. Streptomycin alone could be considered poor.

TABLE IV

Case No.	Aerobic	Anaerobic	Organism	Penicillin	Streptomycin	Aureomycin	Terramycin	Achromycin®	Chloromycetin	Bacitracin
15.	x	..	Alpha streptococcus	−*	−	+†	+	..	+	−
	..	x	Bacteroids; alpha streptococcus	...	..	...	...	..	..	..
16.	x	..	Micrococcus pyogenes var. aureus (coagulase positive)	+	−	+	+	..	+	+
	..	x	No growth	...	..	...	...	..	..	..
17.	x	x	No growth	...	..	...	...	..	..	..
18.	x	..	Beta streptococcus	−	−	−	−	..	−	−
	x	..	Alpha streptococcus	...	..	...	...	..	..	..
	..	x	Bacteroids (gram-negative)	−	−	−	−	..	−	−
	..	x	Diphtheroids (non-hemolytic)	+	−	+	−	..	+	−
20.	x	..	Alpha and beta streptococcus	...	..	...	...	..	..	..
	..	x	Diphtheroid and bacteroids (gram-negative)	...	..	...	...	..	..	..
22.	x	..	M. pyogenes var. aureus	...	..	...	...	..	..	..
	..	x	Aerobacter aerogenes	...	..	...	...	..	..	..
	..	x	Alpha streptococcus; anaerobic micrococcus	...	..	...	...	..	..	..
23.	x	..	M. pyogenes var. aureus (coagulase positive)	+	+	+	+	+	..	+
	x	..	Beta streptococcus	+	+	+	+	+	..	+
	..	x	No growth	...	..	...	...	..	..	..
24.	x	..	Beta streptococcus	+	+	+	..	..	+	+
	..	x	Anaerobic micrococcus	+	−	+	+	..	+	+
29.	x	..	Alpha streptococcus	...	..	...	...	..	..	..
	..	x	No growth	...	..	...	...	..	..	..
35.	x	x	No growth	...	..	...	...	..	..	..
36.	x	x	No growth	...	..	...	...	..	..	..

* − = resistant.
† + = sensitive.

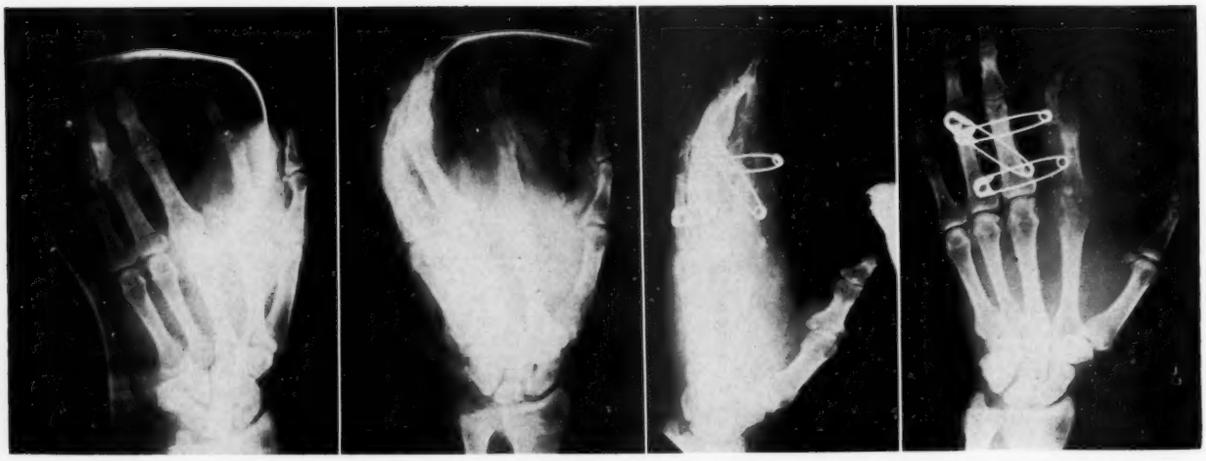

FIG. 1. Case 15. Twenty days after initial wound, January 22, 1953.

FIG. 2. Same case. Thirty-two days after original injury, February 3, 1953.

TREATMENT

In the human bite seen early, thorough cleansing with soap and water is desirable. The wound should be inspected without probing. Limited débridement should be performed. No attempt is made to repair lacerated tendons, and the wound itself should not be sutured primarily. Thorough irrigation with saline is desirable. Resting the hand on a metal splint with a continuous moist saline dressing works very well to minimize infection. If at all feasible the patient should be hospitalized; but since this is almost impossible to do routinely, continuous moist dressings at home may suffice. Penicillin therapy is instituted. If any pus is encountered, cultures should be done immediately and the organism and sensitivity determined.

In some bites that are not too deep thorough washing with soap and water appears to be satisfactory. Tetanus antitoxin is not given.

In cases that are seen late, which is often the case, hospitalization and adequate incision and drainage should be carried out immediately. The pus is cultured; the organisms and sensitivities to antibiotics are determined. The hand is dressed in a metal splint, and continuous soaks and antibiotics are started immediately. The patient is kept in the hospital until the wound is clean and then he is followed up in the out patient department. X-rays are obtained routinely in the late cases. Human bites on other parts of the body may be débrided and sutured primarily.

CASE REPORTS

Several cases are presented in detail to illustrate special features which have been observed previously or which are common in these lesions. Case 22 bears some resemblance to the case reported by Andreasen.[20]

CASE 15. A fifty-five year old white man suffered a bite wound of his left index finger on December 2, 1952. He received no treatment until he was seen on December 20th and admitted to the hospital. At admission, infection had progressed to require incision and drainage of both lateral aspects of the index finger and the mid-palmar compartment. After operation he was treated with penicillin 600,000 units two times a day, streptomycin 0.5 gm. two times a day and moist dressings. On January 30, 1953, extension of the first incision and drainage was done. On the same day treatment was changed to terramycin 5.0 gm. every six hours (culture had shown the organism to be resistant to both penicillin and streptomycin). On February 17th amputation was performed. The skin flaps were left open and appeared to be healing in cleanly when the patient was discharged on February 21st. Figures 1 and 2 show the x-ray progress of this case.

CASE 16. A forty-three year old Negro woman suffered a bite wound of the left index finger at the distal interphalangeal joint about June 14, 1953. She was not seen at the Detroit Receiving Hospital until July 13th, when she had extensive swelling and inflammation. On admission incision and drainage was done, and postoperatively therapy with penicillin 600,000 units two times a day and streptomycin 0.5 gm. every eight hours was started. In addition, warm soapy hand baths four times daily were instituted. Figures 3 and 4 show x-ray progress in this case. Figure 4 shows the

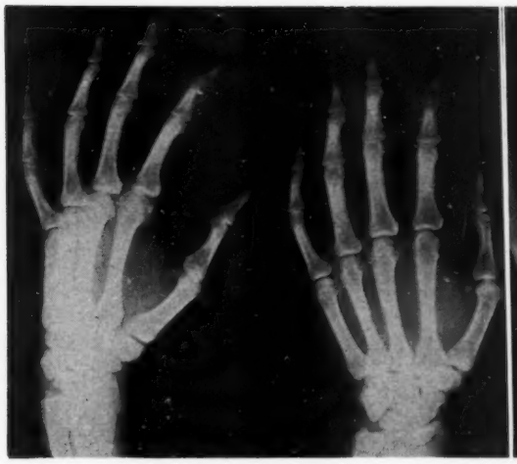

FIG. 3. Case 16. Approximately one month after bite injury, July 13, 1953.

FIG. 4. Same case. Approximately two years after bite injury, May 30, 1955.

FIG. 5. Case 22. Nine days after injury, January 1, 1954.

FIG. 6. Same case. Twenty days after injury, January 12, 1954.

FIG. 7. Same case. Thirty-four days after injury, January 26, 1954.

x-ray taken at follow-up on May 30, 1955. The finger at this time was well healed with no evidence of inflammation; however, there was complete ankylosis of the distal interphalangeal joint.

CASE 22. A forty-nine year old white man received a bite wound of the fifth digit of his right hand on December 23, 1953. When admitted on January 1, 1954, he had extensive cellulitis over the dorsum of the right hand. He was treated with penicillin 600,000 units two times a day, streptomycin 0.5 gm. two times a day and wet dressings. The hand showed outward improvement until January 15th. Because an x-ray taken on January 12th was diagnosed as showing osteomyelitis, the hand was incised over the fifth metacarpal head on January 15th and the bone lesion curetted. The hand was treated by soaking in soapy water and the patient was discharged on January 27th. When the patient was seen on February 9th, the wound appeared well healed with no evidence of inflammation. The x-ray course of the bone infection is illustrated in Figures 5 to 7.

CASE 28. On November 29, 1954, a thirty-eight year old Negro injured his knuckle when he struck a friend in the teeth. The patient was seen at the Detroit Receiving Hospital and received a single injection of penicillin and streptomycin. He did not come in for further antibiotics, nor did he soak his hand in soapy water as advised. When next seen on January 4, 1955, he had considerable swelling and lymphangitis up his arm. Figure 8 shows the appearance of the wound on that day. The patient was treated with penicillin 600,000 units every six hours, streptomycin 0.5 gm. every twelve hours and wet dressings. He was discharged as improved on January 6th to be followed up as an outpatient. On January 11th the inflammation was almost entirely gone. The patient did not keep further appointments. This case shows the rather typical picture of the blow wound.

CASE 32. A fourteen year old Negro girl received bites of both cheeks and was seen the following day with early inflammation. She was treated with penicillin 600,000 units and streptomycin

FIG. 8. Case 28. Film taken on January 4, 1955.

FIG. 9. Case 34. Human bite, distal phalanx of the right index finger.

FIG. 10. Case 36. Three day old infected bite of left thumb.

0.5 gm. daily. She was seen five days later with clean, healed wounds.

CASE 34. A forty-seven year old Negro woman suffered a complete amputation of the distal 4 mm. of her right index finger, including a portion of the nail, on March 14, 1955. She was seen the same day and treated with penicillin 600,000 units and streptomycin 0.5 gm. daily for six days. She also used soaks in soapy water four times a day until March 29th, when she was seen in the clinic with a dry wound which had slight swelling and redness. When seen again on April 5th the wound was still open but was closing and showed no inflammation. (Fig. 9.)

CASE 36. A forty-one year old Negro man received a blow injury to the dorsum of his left thumb on March 25, 1955. On March 28th he came to the emergency room for the first time because of moderate inflammation of the wound. X-ray taken at this time revealed a concomitant fracture of the proximal phalanx. He was treated with penicillin 600,000 units and streptomycin 0.5 gm. daily, with soaks in soapy water four times a day. He was seen in the clinic on April 1st with a decrease in tissue inflammatory signs but pus in the wound. In spite of the fact that the patient thought he needed no further care or hospitalization, we believe time will lead to his eventual reappearance and admission with osteomyelitis. (Fig. 10.)

SUMMARY

1. A brief review of the literature on human bites is presented, with special notice of literature of the antibiotic era.

2. Forty-four cases of human bites seen at the Detroit Receiving Hospital are presented and data tabulated. Seven cases are presented in more detail, with x-rays and photographs.

3. Cultures on eight cases with sensitivities on five of these are tabulated.

4. Discussion of trends and special features of the series is presented.

5. An outline of treatment based on our observations is given.

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Injuries to the Liver and Biliary Tract

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AN ever increasing number of automobiles traveling at greater speeds than ever before accounts for the more frequent occurrence of highway accidents which result in serious injuries. Among the victims of such accidents are persons who sustain major trauma to the abdomen without a penetrating or open wound. A very high mortality rate is associated with injuries to the liver and biliary system in this type of trauma. People in automobile accidents who sustain such massive injury often die from hemorrhage within a matter of minutes. Victims of lesser trauma may reach a hospital where the extent of injury and treatment provided determine whether or not they will survive. The purpose of this article is to discuss injuries to the liver and biliary system which fall into the latter category.

INCIDENCE

Although we do not have at hand actual data from significant series of cases to be of statistical value, we believe from a review of autopsy reports over the past five years that injuries to the liver and biliary system form a major part of deaths in automobile accidents. It has been observed also that the more extensive the injuries to these structures, the more likely is there to be associated injuries of significant magnitude of other parts of the body. Based on a review of the literature[3,4,10] and our observations, we estimate that of those persons in automobile accidents in whom injury to the liver and biliary system from non-penetrating trauma is sustained, one-third will die at the site of the accident, one-third will die within six hours, and one-third will survive.

Non-penetrating trauma which produces rupture of a viscus may cause little evidence of external injury. When rapidly moving automobiles suddenly collide, occupants of the car may sustain injuries to the viscera with little evidence of external trauma, particularly if they are fully clothed. The liver, under these circumstances, is frequently injured. Its location and attachment which prevents its escape from the forces involved, as well as its weight, consistency and shape, are factors contributing to the vulnerability of the liver.

DIAGNOSIS

On examination of any victim of an automobile accident the possibility of injury to the liver and bile ducts should be considered. Such precaution might aid considerably in avoiding the oversight of these injuries until delayed hemorrhage or bile peritonitis is evident. For those patients who sustain massive liver injuries, little can be done since death usually occurs within a matter of minutes and often at the site of the accident. Efforts should be directed toward saving patients whose injuries are of a lesser magnitude, not immediately fatal, but which often result in death within forty-eight hours after the accident. This group, for the most part, comprises injuries which (1) immediately result in some hemorrhage that stops either because of a slight fall in blood pressure or because of normal clot formation within the liver wound; (2) injuries which in addition to hemorrhage are accompanied with escape of bile from the surface of the liver wound which tends to separate the clot; and (3) injuries which have an associated impairment of liver function of varying degree which may have significant effects upon the local and systemic reaction to the injury. This group of patients provides an opportunity for the surgeon to take direct steps toward preventing and/or controlling hemorrhage, and for providing an external escape for bile. At the same time further disturbances of liver function may be averted.

EXAMINATION OF PATIENTS SUSPECTED OF HAVING LIVER INJURY

A complete physical examination to determine the presence or absence of evidence of other injuries and at the same time evaluate the patient's general status is fundamental.

In seeking signs of liver injury pain is the most common manifestation in the conscious patient. This is difficult to evaluate in cases in which there are multiple injuries and is lacking, of course, in unconscious patients. In the first case, pain is present and persistent in the upper abdomen and usually more marked in one of the upper quadrants. The pain increases with movement; often strikingly so on attempted deep inspiration. Radiation of pain to the shoulder on the side of injury is almost the rule if the superior surface of either lobe has been ruptured. Laceration and separation of the inferior surface only is infrequent but may occur without causing diaphragmatic irritation. The escape of blood into the peritoneal cavity is associated with pain and peritoneal irritation which produces easily elicitable signs such as localized tenderness, rebound phenomenon to the point of release and sometimes muscle guarding and rigidity of the abdominal wall. Nausea and vomiting are not constant accompaniments particularly in the early period after injury. Tenderness on compressing the lower portion of the rib cage on the right, plus pain referred to the right shoulder in the absence of rib fractures, is so constant a finding in rupture of the superior surface of the right lobe that these findings alone following an automobile accident constitute ample evidence for the diagnosis of rupture or laceration of the right lobe of the liver. The appearance of a mass in the right upper quadrant caused by either displacement of the liver downward from an expanding accumulation of clot between the diaphragm and the liver, or the appearance of a mass due to hemorrhage in the subhepatic area are commonly associated with shock. Thus they too are unmistakable signs of advanced and serious liver fracture.

The response to injury to the liver is to a degree dependent upon the age and over-all physical condition of the patient. The young and robust amidst the excitement of an accident that may involve others apparently more seriously injured or dead may be unaware of their own injuries and overlook significant complaints. Such patients after being temporarily "knocked out" for a few minutes, sustaining an injury to the liver, may consider the pain which accompanies it as being due to a minor bruise. Hence another reason for careful examination of all persons involved in automobile accidents. Such evaluation should establish first whether or not the patient is in a state of shock and, if not, has he been and has he temporarily recovered, or is he in early and incipient shock. Evidence for blood loss should be sought and specific attention directed toward the liver and biliary system in an attempt to determine local injury with hemorrhage and escape of bile into the peritoneal cavity.

A degree of liver injury sufficient to produce shock usually will result in considerable hemorrhage and some escape of bile. However, it may be that injury to other parts of the body caused the shock and the liver injury may have been sufficient to have resulted immediately in blood loss or bile escape. Thus the shock may be temporary and by the time the patient arrives at a hospital he may have a stable blood pressure and pulse. In either of these two situations hemorrhage may again begin and continue. This together with the escape of bile into the peritoneal cavity can result in rapidly progressive shock which within a short time is irreversible. The patient who is in this category will gain the most from a careful search for evidence of liver injury. If there has been a liver injury of either of the two types described, local findings should enable a proper diagnosis to be made.

Liver injury with hemorrhage and/or escape of bile is accompanied with an increase in pulse rate. The rapidity with which the pulse rate increases is often an index to the magnitude of the injury. An increasing pulse rate is indicative of an approaching fall of blood pressure and shock. We have observed bradycardia in patients in whom bile escapes into the peritoneal cavity both from a liver needle biopsy and following operation upon the biliary tract. Exploration has demonstrated little or no hemorrhage in these patients. Actually we have yet to see a patient with an appreciable amount of blood in the abdomen from a liver injury, with or without a comparable amount of bile, who has not had an increased pulse rate prior to operation.

If the diagnosis of injury to the liver is made or is strongly suspected following such trauma as may be sustained in an automobile accident, the patient should be prepared and operated upon with as little delay as is compatible with anticipating the common exigencies. Delay increases the risk that the injury entails, as well as the risk of the operative procedure.

In those patients in whom the findings are not clear-cut or are minimal but in whom liver injury is strongly suspected either because of the nature of the accident and/or the course of events thereafter, operation will provide the greatest protection. If liver injury is insignificant, the operation may be well tolerated; whereas if such an injury escapes diagnosis because the classic picture is in some part lacking, therapy will be embarked upon too late and the price may be the patient's life.

PREPARATION FOR OPERATION

Over-all Evaluation. This includes a survey of any other injuries the patient has sustained and the possible role they may assume in their relationship to an abdominal operation. It also provides for an estimation of the status of the person's various systems. For example, in surveying the cardiovascular system, a few questions are pertinent. If the patient is young, is there a congenital defect present; if he is in the older age group, is there myocardial disease or advanced arteriosclerosis? Has the patient chronic pulmonary disease or has there been a recent acute infection? All systems should be quickly and efficiently considered in an effort to enable one to anticipate complications and use measures to prevent them.

Correction of Conditions Relative to Injury. If there has been hemorrhage and reduction of blood volume, its correction is indicated. Fluids such as glucose in water and saline may be used first because of their availability, followed by plasma and blood. At the same time parenteral chemotherapy should be instituted. There is ample evidence from both investigative and clinical experience to justify the routine use of antibiotics in trauma to the liver.

Particular attention to the airways of patients who have been in accidents is mandatory before embarking upon an operative procedure or administration of anesthesia.

Decompression of Upper Gastrointestinal Tract. Individuals injured in automobile accidents seldom have an entirely empty stomach. Frequently they have been overindulging in both food and drink. Following injury some will vomit and partially or completely empty their stomach. On the other hand injury is often followed by failure of the stomach to empty itself into the duodenum with resulting stasis of the gastric contents. If the stomach is incompletely emptied, then retained gastric contents become a hazard during anesthesia and operation. All patients who have been in an accident should have their stomachs emptied and lavaged carefully before surgery. Furthermore the maintenance of an indwelling gastric tube to keep the stomach decompressed during and after the operation is a sound surgical practice.

A Large Reserve of Blood Available for Administration Under Pressure If Needed. A patient with an extensive liver injury who has been in shock and recovered as well as the one who may be on the verge of shock can rapidly pass into that state upon the induction of anesthesia followed by the relaxation of the abdominal muscles or the actual opening of the abdomen with even minimal disturbance of its viscera. Because hemorrhage is the greatest single factor contributing to shock in either of these situations blood replacement is our most effective means of correcting it. Before beginning anesthesia and operation it is well to have on hand a supply of blood in excess of the probable normal blood volume. This amount may not be required; but if during the procedure difficulty arises in controlling the hemorrhage from the liver, 4,000 or 5,000 cc. may be used quickly and even more may be required. Auto-transfusion frequently mentioned and occasionally used has little place during operation upon these patients. Anticipation and preparation for such situations will do much to obviate sudden collapse which rapidly becomes irreversible.

Anesthesia. Adequate anesthesia preceded by the least possible disturbance during induction may mean the difference of life and death to the patient with a large wound of the liver when bleeding has almost ceased. A stormy induction may be accompanied with displacement of the clot and a sudden increase in bleeding that may place the patient in irreversible shock before the liver can be exposed. It is likewise important that the anesthesia be such as to permit relaxation so that on opening the abdomen there is no tendency for the viscera to be dislocated suddenly.

OPERATION

Supported by good anesthesia and an adequate reserve of blood that can be given rapidly under pressure, the surgeon's efforts will be facilitated by a generous incision after first

making a small opening in the peritoneum for a preliminary survey. Temporary compression of the portal vein, if there is active bleeding from the liver, may serve a useful purpose while the liver and biliary system as well as abdominal contents are being examined. There are all manner and variations of the extent and nature of these injuries. Indeed no two are alike. The approach to these wounds therefore must be variable. In general it may be said that free and partially detached fragments should be removed. In cases in which the injury is limited to the left lobe, extensive lobectomy may be the procedure of choice. Rupture and superficial lacerations may be closed with any one of the various suture methods described in recent surgical papers on this subject. Where there is complete rupture through capsule and parenchyma after removing free fragments of liver, massive sutures that seek to control hemorrhage and occlude the biliary ducts may be effectual. Absorbable hemostatic sponge material of various types is of great assistance in this attempt. All traumatic wounds of the liver and biliary system should be provided with drainage that will permit the escape of bile through the abdominal wall to the exterior. This is readily accomplished by packing open wounds lightly and by placing rubber-covered gauze drains over closed wounds.

A gallbladder which is lacerated or detached from the liver bed should be excised. Lacerations of the extrahepatic ductal system should be meticulously repaired with five interrupted silk sutures and proximal decompression instituted by means of a rubber T-tube. Following cholecystectomy or repair of part of the extrahepatic ductal system, there should be liberal provision for external drainage.

Following operation for trauma to the liver all patients will have some degree of peritonitis due to bile, hemorrhage or infection. Their treatment should include all measures commonly employed including gastrointestinal decompression, maintenance of proper water and electrolyte requirements, and chemotherapy. We are well aware that even after the successful accomplishment of these measures some patients do poorly and fail to recover. This group of patients have usually sustained some interruption of the many normal physiologic functions of the liver.

When we consider the numerous functions of the liver and their complexity and at the same time remind ourselves that we only partially understand their relation to the total body economy, we realize that these aspects of liver injury have as yet received insufficient attention.

We may emphasize this situation by comparison of an injury to an extremity, such as a fracture of the femur, with a rupture of the liver. In the former there is pain, perhaps some hemorrhage and the breakdown of surrounding tissue. If the fracture is immobilized, the pain is decreased and the breakdown of surrounding tissues may be so minimal that there will be little systemic disturbance. In rupture or fracture of the liver not only are we less able to immobilize it well in an attempt to prevent further hemorrhage and escape of bile but also we have varying degrees of interruption of the normal functions of the liver.

We may outline them much as the physiologist does to indicate how far reaching their derangements may extend. These include: (1) formation of (*a*) fibrinogen, (*b*) prothrombin, (*c*) heparin; (2) metabolism of (*a*) carbohydrate, (*b*) protein, (*c*) fat and (*d*) vitamin; (3) bile production (*a*) excretion of bilirubin, (*b*) excretion of cholesterol and (*c*) formation and excretion of bile salts; (4) detoxication (*a*) bacteria, (*b*) noxious products of normal metabolism and (*c*) excretion of certain ingested injurious materials; (5) hematinic principle production; and (6) liberation of vasodepressor material and antidiuretic principle.

These are but a partial list since it includes only those functions that are more commonly described. However, they alone are sufficient to illustrate the multiple processes that go on in the liver. Most of them are essential to life. All of them are relative to the body economy as a whole and hence their derangement, which at any time would be detrimental, may become catastrophic when associated with hemorrhage, shock, peritonitis, or other significant trauma. For example, interference or failure in the synthesis of prothrombin is always of concern because of the resulting bleeding tendency. When there has been massive liver injury with rupture and hemorrhage that has ceased because of the normal clotting mechanism as the blood pressure fell, then a lack of prothrombin becomes a far more serious matter since renewed bleeding can quickly result in death. Other liver functions over a longer period of

time may play an equally important role. Their preservation, therefore, becomes as major an objective in any operation upon the patient with an injured liver as does the more obvious, such as control of hemorrhage and diversion of escaping bile.

A patient who has sustained major trauma in an accident of any kind should be considered as possibly having liver damage even though there is no external evidence of such. When liver injury is suspected, patients should be operated upon as soon as their general condition will permit. Examination of the liver should include careful inspection of its entire surface for evidence of laceration, and in addition careful examination of evidence of subcapsular injury. Needle aspiration of the liver is justified although this is not generally satisfactory. The removal of devitalized necrotic or sequestrated liver tissue is essential. When the trauma has produced a capsular laceration or when the surgeon has had to transect the liver surface in order to evacuate traumatized liver tissue, or when a cholecystectomy or repair of the extrahepatic ductal system has been done, it is most important that biliary drainage[5,7] from such an area be diverted to the exterior by the use of a drain. Accessories to the control of hemorrhage are to be found in hemostatic aids and multiple ligatures. The mortality in massive trauma to the liver is high and justifies our seeking ways and means of combatting the derangements of the normal physiologic functions of the liver.

COMMENTS AND CONCLUSION

Death following injury to the liver and bile ducts may be caused by hemorrhage, shock, biliary drainage, impairment of liver function, necrosis or infection. It may be due to any one of these or to any combination of them. The first two, hemorrhage and shock, are by far the most frequent causes of early death. The immediate control of all of these complications are important in the care of the patient. It is our belief that this can best be accomplished by prompt surgical measures that include supportive therapy before, during and after operation that is directed at the control of hemorrhage, the diversion of escaping bile, the removal of detached liver fragments and the prevention of infection. As a result of experience in recent wars and in civilian practice open and penetrating injuries to the liver and bile ducts are accorded early and direct surgical management.

Sparkman and Fogelman,[8] in a review on wounds of the liver of all types including both penetrating and non-penetrating, collected reports on 1,245 patients with a mortality ranging from 27 to 81 per cent. He stressed that two groups were treated with early operation. First were Army patients of World War II who had the lowest death rate (27 per cent, 829 patients), and second were those from the Parkland City County Hospital in Dallas, Texas (100 patients, with a 10 per cent mortality). He believed this was in contrast to the therapy accorded those in the older case series reported.

For many years there has been a widely advocated concept that those with liver injuries from non-penetrating injuries are best treated by "conservative" or non-operative therapy. Hinton[2] in 1929 wrote, "In cases of intraabdominal hemorrhage if the hemorrhage is due to a ruptured liver, conservative treatment should be followed." In 1931 Robertson[6] reporting the experience of a group in Sayre, Pennsylvania, expressed a somewhat similar attitude. Allen[1] in discussing his experience with a small series of liver wounds in 1930 advocated operation. A similar attitude was evident in a report on this matter by Krieg[3] from Detroit in 1936. Welch and Giddings[9] (1950) in a review of 200 consecutive cases of abdominal trauma, about 10 per cent having major liver trauma, were convinced that early operation would best protect the patient.

In reviewing case series of liver injuries, the result of non-penetrating trauma, it is evident that patients with massive involvement have a high mortality rate regardless of the therapy accorded them. It is difficult to determine when surgical operation has saved life. The entire clinical picture is often complex and associated with other injuries. However in our own experience we are quite confident that severe hemorrhage which would have been fatal has been controlled. Furthermore that bile escaping from both intrahepatic and extrahepatic ducts has been guided to a proper channel, or to the exterior, and has been diverted from the peritoneal cavity. Fragments of detached liver have been removed, with survival of patients.

The attitude toward the management of

patients with possible liver and biliary tract injuries is of paramount importance. If the approach includes a suspicion of liver injury in patients who have been in automobile collisions and similar accidents, it is less likely that these injuries will be overlooked. This is in contrast to an expectant approach in which the patient is operated upon after the clinical picture of liver injury and its complications have developed; thus the period of greatest opportunity to control the factors described as the more common cause of death has been lost. This is applicable in particular to those persons who do not die soon after injury and arrive at our hospitals still in a retrievable state. For these patients an early direct surgical approach, being ever mindful of the disturbances that trauma may cause to the normal physiologic function of the liver, should be beneficial.

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Hematoma of the Abdominal Wall

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HEMATOMA of the abdominal wall, an uncommon clinical entity, may be mistaken for any of the myriad intra-abdominal and abdominal wall conditions which are responsible for an acute surgical condition of the abdomen. In reporting on such a hematoma others have used the terms spontaneous hematoma of the abdominal wall, rupture of the epigastric artery, rupture of the rectus abdominis muscle and hematoma of the rectus abdominis muscle. By recognizing such a hematoma early, much effort and numerous time-consuming laboratory procedures employed in the diagnosis of the acute abdominal condition may be avoided; immediate therapy which is relatively simple may be instituted and the duration of the patient's hospital stay may be lessened.

Even though hematoma of the abdominal wall is not diagnosed correctly before operation in a high percentage of cases, this condition has been recognized and accurately described for a long period of time. Hippocrates and Galen presented correct descriptions of the condition.[48] Maydl[45] referred to Leonardo da Vinci's cases previous to 1519; he reviewed the literature from 1809 to 1880 and collected fourteen cases of spontaneous rupture of the epigastric artery reported before 1880. In 1857 Richardson[50] described a case in which a healthy, young adult white male suffered hemorrhage in the abdominal wall after vigorous exercise. In 1923 Wohlgemuth[69] collected 127 cases from the literature, 107 of which were traumatic due to internal violence. In 1930 Herlyn[25] compiled statistics on forty-three cases in the literature and emphasized the inaccuracy in preoperative diagnosis. In 1938 Payne[48] found in the literature the records of 165 cases for analysis of the histories, operative findings and pathologic investigations. This present report summarizes data on seventy-nine cases initially reported in the literature since Payne's report in 1938, and presents data on five additional cases to make a total of eighty-four. Three of these cases were seen in private practice and two on the clinical service of the Ohio State University Hospital.

Anatomic Considerations. In this condition the most important structures involved are the rectus abdominis muscle and its sheaths, the epigastric vessels and the semi-circular line of Douglas. Cullen and Brödel[13] described the long, narrow belly of the rectus abdominis muscle as made up in reality of separate muscles that act as one because of the tendinous bands that run transversely. It can expand and contract as much as 9 fingerbreadths. Terrific pressure is exerted in the act of defecation, straightening up or violent coughing spasms. Running beneath this muscle in loose areolar tissue are the inferior epigastric artery and the superior epigastric artery—a long anastomosis between the external iliac artery inferiorly and the internal mammary artery superiorly. Lying posterior to this anastomosis from the thoracic cage to the semi-circular line of Douglas there is the posterior rectus sheath; inferior to this line there is no posterior rectus sheath. The inferior epigastric vessel sends many branches to the rectus abdominis muscle. The proximity to the peritoneum, especially below the umbilicus, probably causes the signs of peritoneal irritation so often observed when the vessel bleeds. In the exceptional case, bleeding may extend as far superiorly as the thoracic cage and as far inferiorly as the perivesical space. Because there is no posterior sheath inferior to the semi-circular line of Douglas, a hematoma may extend directly posteriorly and, on bimanual pelvic examination, simulate a pelvic mass, as an ovarian cyst.

Etiologic Factors. There has been much speculation as to the exact etiology of hematoma of the abdominal wall and as to its predisposing and exciting factors. Conclusive evidence is still lacking, and the cause of the hematoma is usually a matter of speculation and assumption. In many cases associated conditions which seem to favor the formation of

the hematoma can be pointed out. In some of the cases which have been described as spontaneous hematoma, careful history will elicit one of the factors listed in Table 1; but in other cases no discernible factor will be indicated. In every case, however, a definite endeavor should be made to determine a possible cause, for occasionally there may be a medical, legal or industrial reason to prove or disprove a possible cause of hematoma.[12,47,69]

Trauma or violence may be responsible in a variety of ways for hematoma of the abdominal wall. As pointed out by Cullen and Brödel,[13] there may be excessive change in length between maximum contraction and stretching of the lower rectus muscle, and such change in muscle length will also be accompanied by change in length of the epigastric vessels and possible rupture of such vessels. In the various types of internal or indirect trauma listed in Table 1, such as severe coughing or sudden exertion in work or exercise, there may be such changes in length of the rectus muscle. Direct blows on the abdomen or repeated paracenteses may be responsible for a hematoma. Operative injury to the inferior epigastric vessels may result in the development of a palpable hematoma as late as two weeks[31] or even as late as a month after laparotomy.[47]

Murray and Burger[47] reported a case in which a hematoma developed after dicumarol therapy for acute thrombophlebitis of the leg. They note that "With no history of strain or trauma, it was our opinion that anticoagulant therapy superimposed on sclerosis in a hypertensive inferior epigastric vessel was the main cause of the apparent spontaneous hemorrhage. Slight exertion, of course, may have initiated the process." Carp[9] reported a similar result with dicumarol.

Prior disease, such as typhoid fever, typhus fever or other severe infectious diseases, may be a predisposing factor to the development of a hematoma of the abdominal wall. Cullen and Brödel[13] emphasized the greater frequency with which hyaline degeneration of muscle fibers follows acute infectious diseases in the rectus abdominis than in other muscles of the body, and stated that such hyaline degeneration makes the rectus abdominis muscle weaker so that it ruptures with the slightest physical exertion.

Diseases occurring simultaneously with the hematoma may be regarded as predisposing or exciting factors. Such diseases may exert their influence by being responsible for a direct muscle strain, as the coughing associated with respiratory tract infections. Other diseases in this group may produce their effect directly on the individual muscle cells, as in miliary tuberculosis or chronic alcoholism. Finally, in this group, the weakening of the arteries that occurs in the arteriosclerosis associated with advanced age must be considered.

Several series of cases[14,34,40,62] have been reported in which hematomas of various sizes have occurred during pregnancy, labor and the puerperium. At these three times there is unusual tension on the rectus abdominis muscles of a woman.

In some cases[19,48,49] no discernible factor

TABLE 1

PREDISPOSING AND EXCITING ETIOLOGIC FACTORS FOR HEMATOMA OF ABDOMINAL WALL

- Trauma or violence:
  - Internal:
    - Physical exertion in work or exercise[4,22,26,33,42,66,68,69]
    - Twisting[51]
    - Vomiting[19]
    - Defecation (Case III)
    - Cough (Cases IV and V)[5]
    - Tetanus[27]
  - External:
    - Blow on abdomen[2,24,30,70]
    - Laparotomy[31,47]
    - Paracentesis (Case II)
- Anticoagulants (Case I)[9,47]
- Diseases which have occurred before hematoma developed:
  - Typhoid fever (Cases II and III)
  - Typhus fever[27]
  - Miliary tuberculosis[27]
- Diseases present at time hematoma developed:
  - Asthma[60]
  - Acute coryza[3]
  - Bronchopneumonia[67]
  - Influenza[47]
  - Acute bronchitis (Case IV)[1,11]
  - Whooping cough[5,39]
  - Arteriosclerotic and hypertensive cardiovascular disease (Case V)[10,16]
  - (?) Epigastric aneurysm[64]
  - Blood dyscrasias[52,64]
  - Miliary tuberculosis[18]
  - Typhus fever[18]
  - Chronic alcoholism[70]
  - Tetanus[27]
  - Burns[60]
  - Typhoid fever[60]
- Pregnancy, labor and puerperium[14,34,40,62]
- No discernible factor[19,48,49]

can be demonstrated. The patient suddenly and unexpectedly notices a mass in the abdominal wall. These are the truly spontaneous hematomas.

In the five cases initially described in this paper a variety of factors were considered to be responsible for the development of the hematoma. (Table II.) In Case III strain associated with defecation and in Cases IV and V the strain associated with repeated bouts of coughing were thought to be exciting factors. In Case II the trauma from repeated paracenteses appeared to be a possible etiologic factor. In Case I no history of strain or trauma could be elicited. It seems significant, however, that in this case the patient had been given heparin to treat acute thrombophlebitis. Several days after the administration of the first dose of heparin multiple subcutaneous ecchymoses developed as well as a mass in the right upper quadrant which was found to be a hematoma.

TABLE II
DATA ON FIVE CASES OF HEMATOMA OF ABDOMINAL WALL

Case No.	Age, Sex and Race	Etiologic Factors	History of Typhoid Fever
I	71, F, W	Heparin	No
II	82, M, W	Paracenteses; cirrhosis with ascites	Yes
III	54, M, W	Defecation	Yes
IV	62, F, W	Cough; bronchitis	No
V	41, F, W	Cough; hypertensive cardiovascular disease (decompensation)	No

In two of the five cases a definite history of typhoid fever was given by the patient. In both instances the typhoid fever had occurred years prior to the development of the hematoma. In Case III, in which hematoma developed at the time of defecation, typhoid fever had occurred thirty-three years previously; in the case in which paracenteses had been done, typhoid fever had occurred eight years previously.

Incidence. Hematoma of the abdominal wall is a condition which is not too frequently seen. Although a number of papers have appeared on this subject, usually only one or two new cases are reported in each paper.

The age at which the condition occurs is determined to a great extent by the etiologic factor involved; for example, the hematomas seen in pregnant women occur in the younger adult age group. In the eighty-four cases reviewed since 1939 the age range was nineteen to eighty-six. The sex is usually female, 59 per cent of the hematomas occurring in women. The race involved is almost always white. Only two cases were listed as being in Negroes; however, the race of the patient is frequently not given in the literature.

TABLE III
SIGNIFICANT FINDINGS IN FIVE CASES REPORTED HEREIN

Case No.	Duration Preoperatively	Pain	Location of Mass	Tenderness
I	7 days	Yes	R.U.Q.	Yes
II	About 2 wk.	No	R.L.Q.	Yes
III	6 hr.	Yes	R.U.Q.	Yes
IV	5 days	Yes	R.L.Q.	Yes
V	1 day	Yes	L.L.Q. and L.U.Q.	Yes

Clinical Picture. The intensity and rapidity of onset is usually proportional to the size of the hematoma and the amount of irritation produced on the peritoneum of the anterior abdominal wall. In the typical case the patient notices a sudden onset of sharp, moderate to severe pain which is localized in one quadrant of the abdominal wall and which may occur at any time during the day or night. Some patients have stated that they have felt as though something had snapped in the abdomen. After the onset of pain the patient frequently loses his appetite; and if the hematoma is large enough, he may begin to vomit. Diarrhea does not occur but constipation may develop if there is considerable peritoneal irritation. In four of the five cases presented in this paper, as seen in Table III, the patient did experience pain; an eighty-two year old man who had cirrhosis of the liver with ascites, and in whom hematoma developed after multiple paracenteses, did not complain of pain. The duration between the time of onset of pain and the time of operation is usually determined by the severity of the pain and the surgeon's awareness of the possibility of a hematoma. In the five cases, as shown in Table III, the duration preoperatively varied from six hours in a

middle-aged white man, who was considered to have an incarcerated hernia, to about two weeks in an elderly white man who did not complain of pain.

Usually there is a mild general constitutional reaction with slight to moderate elevation of temperature and pulse; with very large hematomas there may be, however, considerable elevation of pulse and even a fall of the systolic blood pressure to shock levels. As is evident in Table IV, in Cases I and IV there were temperature elevations to 101.2° and 101°F., and pulse rate elevations to 128 and 100, respectively.

TABLE IV
TEMPERATURE, PULSE RATE AND TOTAL WHITE BLOOD CELL COUNT

Case No.	Highest Temperature (°F.)	Highest Pulse Rate	Highest White Blood Count
I	101.2	128	7,650
II	98.6	100	11,250
III	99	100	10,850
IV	101	100	18,850
V	97.8	100	8,000

On inspection of the abdomen a mass may be seen which is localized to one of the quadrants of the abdominal wall. In the eighty-four reviewed cases the hematoma was in the right lower quadrant in 53 per cent, and in the left lower quadrant in 38 per cent. At the time of onset of pain the color of blood is not visible through the superficial fascia and the skin; in cases which have been watched for a number of days ecchymosis may be present, particularly in thin individuals. Of the five cases reported in this paper, only in Case I was there some discoloration of the skin. The hematoma is slightly tender, but only infrequently is there local temperature elevation. Spasm of the adjacent muscles is usually not present. Unless there is a large hematoma with considerable peritoneal irritation, peristaltic sounds are audible. The mass itself is firm but not hard. Because of the restricting posterior rectus sheath above the semi-circular line of Douglas, the hematoma above this line is smaller than one below this line. Because of the absence of the posterior rectus sheath in the lower abdomen, the hematoma may rapidly expand and develop posteriorly and inferiorly into the pelvis, so that on bimanual pelvic examination it is often mistaken for a twisted ovarian cyst or some other pelvic pathologic condition. Rarely the hematoma has ruptured through the peritoneum; if free bleeding occurs into the peritoneal cavity with such rupture, the mass may disappear.[6]

TABLE V
PREOPERATIVE DIAGNOSES IN CASES OF HEMATOMA OF ABDOMINAL WALL REPORTED IN THE LITERATURE

Anterior abdominal wall:
- Incarcerated hernia (Cases III and IV)

Abdomen and pelvis:
- Acute appendicitis[3]
- Appendiceal abscess[29]
- Large bowel obstruction[37]
- Carcinoma of cecum (Case II)
- Carcinoma of colon[26]
- Cancer of rectum[10]
- Intussusception[2]
- Ruptured diverticulitis[38]
- Omental cyst or tumor[46]
- Mesenteric cyst or tumor[60]
- Pancreatic cyst[46]
- Pelvic inflammatory disease with intestinal obstruction[26,35]
- Torsion of ovarian cyst (Case V)[1,59]
- Ectopic pregnancy[65]
- Threatening rupture of uterus[19]
- Torsion of pedunculated leiomyoma uteri[28]
- Tumor of kidney[28]

Laboratory Data. The laboratory has little to offer to aid in the diagnosis of a hematoma of the abdominal wall. If the bleeding is excessive, there will be a corresponding fall in the total erythrocyte count, the hemoglobin and the hematocrit. If there is marked peritoneal inflammation, there may be associated leukocytosis and an increase in the percentage of neutrophils. Leukocytosis, however, may be more an index of the severity of the associated etiologic condition than of the hematoma itself.

On occasion, if the hematoma is quite large, its outline may be seen on roentgenograms of the abdomen. For Case V such an outline may be seen in Fig. 5; preoperatively the roentgenologist called attention to this area which suggested to him a soft tissue mass.

Differential Diagnosis. Hematoma of the abdominal wall has been mistaken for almost every common or uncommon acute condition of the anterior abdominal wall, abdomen and pelvis in which a mass is one of the outstanding characteristics. In Table V are listed the preoperative diagnoses of cases which have been

reported in the literature from 1939 to 1955, inclusive. In 100 cases found in the literature prior to 1946 Teske[61] found that 17 per cent of the cases were diagnosed correctly prior to operation. In the present series of five cases the correct preoperative diagnosis was made in Case 1.

Two signs have been described which may be of value in differential diagnosis. Fothergill[17] noted that if an abdominal mass is palpable with the patient in both the supine and sitting positions and if the mass cannot be moved from side to side, the diagnosis of a hematoma of the abdominal wall must certainly be considered. Romanzew[53] has pointed out that with a hematoma in the rectus abdominis muscle the limits of the swelling correspond with the outline of the sheath of the muscle and that the swelling does not pass beyond the midline or the lateral border of the muscle.

A fairly rapid increase in the size of a superficial abdominal mass is suggestive of an abdominal wall hematoma.

Diagnostic needle aspiration may result in the withdrawal of blood from the hematoma. Such aspiration may be futile, however, since the hemorrhage may be interspersed among the muscle fibers and since the blood may be clotted.

The chief aids in diagnosis are an accurate history, the appearance of a palpable mass which is fixed within the abdominal wall and an alertness to the possible existence of the condition. As Reid[49] and Lisenby[41] have emphasized, the correct preoperative diagnosis depends almost entirely upon the clinician being "hematoma conscious." If he is to recognize the condition preoperatively, he must think of the condition when confronted with a painful mass of sudden formation in the abdomen.

Treatment. When there is severe and moderate bleeding, surgery is necessary to obtain hemostasis at the site of the hematoma. The mild condition may be treated conservatively. Concomitant generalized and local pathologic changes, particularly the etiologic conditions, must be corrected if possible. Blood replacement and general supportive measures may have to be carried out vigorously.

Surgical intervention consists primarily of incision and evacuation of a clot which could be the seed of a chronically painful mass or even of an abscess. Bleeding vessels, of course, are ligated.

The advisability of providing for postoperative drainage will depend upon the extent of the hemorrhage, the amount of hemostasis which can be obtained in the muscle, the size of the dead space produced by the hematoma and the opinion of the individual surgeon. A drain was not used in the five cases reported in this paper. In Case 1 approximately 35 cc. of serosanguineous, thin fluid were aspirated on the twelfth postoperative day even though very meticulous hemostasis with transfixing sutures had been employed at operation. In view of these considerations it is probably best to insert a thin, small Penrose drain down to the dead space created by the evacuation of the hematoma if there is any doubt about the hemostasis at the end of the operation.

In treating hematomas in soldiers who were in training during World War II, Bowers and Richard[4] usually used a conservative plan of absolute bed rest with heat to the affected muscle. By such a plan, however, Vigdoff[66] had one soldier whose convalescent period was ten weeks because of a painful, tender mass.

Operative Findings. At the time of operation the hematoma becomes evident to the surgeon in one of two ways. Either the surgeon makes the incision into the hematoma and recognizes it immediately, or sometimes he incises beyond the hematoma and opens directly into the peritoneal cavity. By being aware of the possibility of a hematoma of the abdominal wall the surgeon can avoid opening the peritoneal cavity.

If the incision is made directly over the mass, a bluish color can be discerned through the anterior sheath of the rectus abdominis muscle. When this sheath is opened, the rectus abdominis muscle may be found to be intact, a few strands may be severed or the ends may be completely separated. The hematoma may be diffusely spread throughout the rectus muscle or it may be a well localized clot. Bleeding arteries or veins may or may not be present. Dwyer,[16] in describing one of his cases, stated that on incision of the sheath of the rectus a blood clot became evident in the substance of the muscle, the fibers of which, although intact for the greater part, were separated into long strands except for what appeared to be the focus of the bleeding where the appearance suggested the destruction of a miniature explosion, with muscle fibers twisted and strewn in all directions.

In many of the cases reported in the literature the operating surgeon did not realize that he was dealing with extraperitoneal hematoma until after he had opened the peritoneal cavity. On exploration no mass was seen deep to the abdominal wall. Careful inspection of the posterior aspect of the abdominal wall, however, showed a well defined mass with a bluish discoloration which, of course, was the posterior wall of the hematoma.

Regarding the question as to whether the muscular or vascular tissues are primarily involved in rectus hematoma, Rogatz and Rubin[52] point out that there is no unanimity of opinion. Some surgeons[18,56] stress rupture of the muscle but others[48,57] describe rupture of the inferior epigastric artery. Smith[58] states that ordinarily the tearing of the rectus abdominis muscle and the rupture of the inferior epigastric vessels or their branches happen simultaneously.

Usually no material is available for pathologic study because in most instances the rectus abdominis muscle is intact or because, even if infiltrated with hematoma or rupture, it has not been removed. In a case reported by Walter and Landesman[67] in which the patient had a cough from bronchopneumonia, the right rectus muscle was friable and necrotic, and the entire muscle was removed. Histologic study of the excised muscle indicated ". . . extensive hemorrhage with beginning of focal organization." Cullen and Brödel[13] observe that "It seems quite clear that Zenker's degeneration of the recti muscles is a not infrequent accompaniment of influenza and influenzal pneumonia and that the muscle fibers become so friable that rupture occurs with varying degrees of hemorrhage."

Mortality Rate. Except for hematoma occurring during pregnancy, deaths from hematoma of the abdominal wall are uncommon. In 1946, in 100 cases collected from the literature and including one case of his own, Teske[61] found a mortality rate of 4 per cent. In the eighty-four cases analyzed in this paper there were four deaths, a mortality rate of 5 per cent.

If deaths occur in non-pregnant patients, they may be more attributable to an associated condition such as decompensated hypertensive cardiovascular disease; in some instances the deaths cannot be explained even after postmortem examination.[10,30,37,44]

Occurrence of hematoma is more serious in pregnant women than in other individuals. Sheehan[55] emphasized the importance of this condition during pregnancy. He stated that "Recent reports show a maternal mortality of 12% and a fetal mortality of 25%." Torpin[65] found a maternal mortality rate of 15 per cent. Thomas[63] noted a maternal mortality rate of 13 per cent and a fetal mortality rate of approximately 50 per cent.

CASE REPORTS

CASE 1. This seventy-one year old white, mentally retarded woman was admitted to the hospital on August 22, 1955, for swelling of the right lower leg which was associated with chills and fever.

As a mentally retarded woman, she had been under the supervision of the Columbus State School for approximately forty-five years. She denied any history of typhoid fever.

On physical examination she appeared to be a fairly well preserved, conscious, mentally retarded, not very well developed white woman who appeared her stated age. The thyroid gland was diffusely enlarged so that it was easily palpable. Fine rales were audible over both lung bases. The heart appeared to be slightly enlarged. The cardiac rhythm was grossly irregular. A systolic murmur was heard at the apex which was transmitted to the left axilla. At the time of admission no abnormal masses were felt in the abdomen. The right lower extremity below the knee was erythematous with multiple petechiae being visible and with 2 plus pretibial and pedal edema. Homans' sign was negative bilaterally. There was necrotic debris from a little blister on the dorsum of the right fourth toe.

Laboratory data were as follows: The erythrocyte count was 3,400,000; hemoglobin, 11.4 gm. per cent; total leukocyte count, 5,150. Findings on urinalysis were not remarkable. The blood urea nitrogen was 19.5 mg. per cent and the fasting blood sugar was 92 mg. per cent.

Fluoroscopic examination indicated generalized cardiac enlargement with left atrial, left ventricular and right ventricular enlargement. An electrocardiogram showed auricular fibrillation, myocardial changes and reduced voltage.

In view of the findings that indicated acute thrombophlebitis in the right lower extremity, the patient was given 300,000 units of procaine penicillin twice a day, 50 mg. crystalline heparin at the time of admission and 575 mg. heparin during a three-day period, so that the Lee-White blood-clotting time was raised from seven minutes to a maximum of twenty-one minutes. In view of cardiac findings the patient was digitalized and maintained on adequate doses of digitalis.

On the afternoon of August 25th the patient was

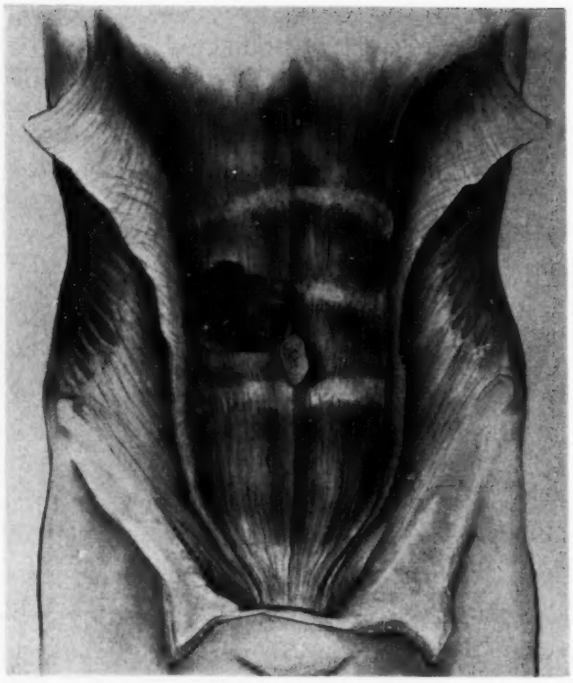

FIG. 1. Location and relative size of hematoma of abdominal wall in Case 1.

found to have a distended abdomen, hypoperistalsis and a slightly tender mass, 7 cm. in diameter, in the right upper quadrant of the abdomen. She was not nauseated and had not vomited. Her temperature was 99.2°F., pulse 120, respirations 28, and the total leukocyte count 6,800.

In addition, it was noticed that the patient had some ecchymoses on her extremities.

In view of these findings the heparin was immediately discontinued. The mass in the right upper quadrant of the abdomen persisted. A cholecystogram on August 31st indicated a visualizing gallbladder. Prior to this x-ray examination, one consultant believed the patient might have hydrops of the gallbladder.

The preoperative diagnosis was hematoma of the right rectus muscle.

Operation was performed on September 2nd. Under pentothal® and endotracheal inhalation anesthesia a slightly oblique, right upper quadrant incision was made over the mass which was located just above the transverse level of the umbilicus and about 3 cm. to the right of the umbilicus. The incision was developed down to the anterior rectus abdominis muscle sheath. As the superficial fascia was dissected off the anterior rectus sheath, there was moderate adherence between the superficial fascia and the anterior rectus sheath which appeared to be bluer then the surrounding tissue. The mass which caused this prominence appeared to be about 7 cm. in diameter. On aspiration with a No. 18 needle nothing could be obtained; on aspiration with a No. 16 needle some very dark blood was withdrawn. A vertical incision was made through the anterior rectus abdominis muscle sheath, and a cavity containing a hematoma 5 cm. in diameter was encountered. This hematoma was removed by blunt dissection with a finger and a bone curet. At no time did any free bleeding occur in the hematoma cavity. This hematoma appeared to lie in the body of the rectus abdominis muscle between the anterior and posterior rectus sheaths. There was complete division of the fibers of the rectus muscle in the transverse plane. The superior epigastric artery was not definitely isolated. The wound was copiously irrigated and closed without a drain. The divided rectus muscle was approximated loosely with No. 00 black silk sutures, the anterior rectus sheath with interrupted No. 00 black silk sutures, and the superficial fascia and skin with two layers of interrupted No. 0000 black silk sutures. The patient appeared to withstand the procedure well. (Fig. 1.)

The patient's postoperative course was excellent except for the aspiration of approximately 35 cc. of a thin, serosanguineous fluid on September 14th.

Final diagnosis: (1) hematoma of the abdominal wall (superior half of the right rectus abdominis muscle); (2) acute thrombophlebitis and cellulitis of the right lower extremity treated with heparin; (3) rheumatic heart disease with mitral insufficiency, auricular fibrillation and mild congestive heart failure; (4) mental deficiency; (5) non-toxic, nodular goiter.

CASE II. This eighty-two year old white man was admitted to the medical service of the hospital on July 27, 1953, for weakness of nine months' duration. Approximately three weeks prior to admission he had fallen, and since that date he had been so weak that he had been unable to walk. No fractures were found on examination.

When he was a child he had had measles, scarlet fever and mumps. In 1946 he had had typhoid fever from which he recovered slowly. For many years he had had an easily reducible, right inguinal hernia.

Examination on admission showed no abnormal findings except for generalized debility and a right inguinal hernia. No abnormal masses were noted in the abdomen.

The patient's course was not remarkable until the middle of May, 1954. At that time it was noted that he had ascites. Attempts at removal of peritoneal fluid by paracenteses on June 12th and 22nd were unsuccessful. The distention persisted.

A few days later a hard mass approximately 10 cm. in diameter was felt in the right lower quadrant. The mass was not adherent to the overlying skin, was somewhat movable, appeared to be in the peritoneal cavity and appeared unrelated

to the right inguinal hernia. The abdomen was still distended. Peristaltic sounds were hyperactive and high pitched.

Laboratory and x-ray studies revealed the following: On June 21st the total erythrocyte count was 4,080,000; hemoglobin, 11.5 gm. per cent; and total leukocyte count, 5,350. On June 25th findings on urinalysis were within normal limits except for 50 mg per cent of albumin.

On July 7th a scout film of the abdomen showed marked gaseous distention of the stomach and gas in both the large and small bowel. Barium enema studies failed to indicate an intrinsic tumor within the colon.

The preoperative diagnosis was (?) carcinoma of the cecum.

Operation was performed on July 13th. Through a lower right paramedian incision, abdominal wall exploration and laparotomy were performed under procaine local, pentothal and inhalation anesthesia. As the incision was being developed, a 10 by 8 by 2 cm. hematoma was encountered which appeared to consist of old blood lying in the substance of the ruptured right rectus muscle. (Fig. 2.)

As the peritoneal cavity was opened, about 2,500 cc. of thin, yellow, ascitic fluid was aspirated. On abdominal exploration the liver was one-half its normal size; on its surface there were numerous small nodules 2 to 4 mm. in diameter. Otherwise the abdominal exploration was not remarkable.

All of the ascitic fluid was removed. The wound was closed without a drain in layers with a continuous, locked No. 1 chromic catgut suture for the peritoneum, posterior rectus sheath and transversalis fascia, and with interrupted silk sutures for the remaining layers.

The patient recovered from the operation and was placed under the care of the medical service again on July 23rd.

Final diagnoses: (1) hematoma of the abdominal wall (inferior half of right rectus abdominis muscle); (2) status post-paracenteses; (3) cirrhosis of liver with ascites; (4) status post-typhoid fever; (5) right indirect inguinal hernia.

Case III. This fifty-four year old white man stated that while straining at stool three hours prior to hospital admission he suddenly experienced crampy pain in the right upper quadrant of his abdomen. Because he could not find relief standing up, sitting or lying down, he called his physician who thought it necessary to administer morphine for the relief of pain and to admit the man immediately to the hospital on August 2, 1946.

At the age of twenty-one, the patient had had typhoid fever.

On the present admission, the patient appeared to be an acutely ill, moderately obese, conscious, white man with a temperature of 99°F. and a pulse

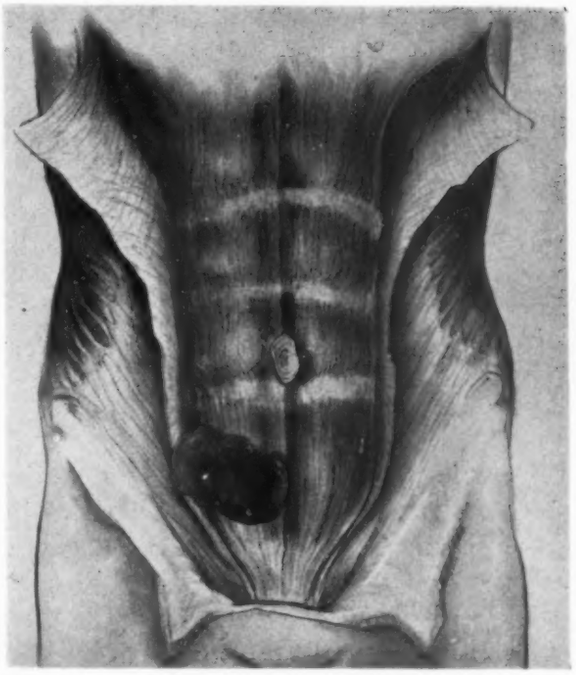

Fig. 2. Location and relative size of hematoma of abdominal wall in Case II.

of 100. In the right upper quadrant of the abdomen a poorly defined tender, non-reducible mass approximately 4 by 6 cm. was palpable. The remainder of the examination was essentially negative.

Laboratory data were as follows: The total leukocyte count was 10,850; the total erythrocyte count and urinalysis were within normal limits.

The preoperative diagnosis was incarcerated ventral hernia.

Approximately six hours after the onset of pain an emergency operation was performed under pontocaine spinal anesthesia. A large hematoma was found between the anterior and posterior rectus abdominis muscle sheaths in the right upper quadrant. The anterior rectus sheath was opened transversely and the clot was evacuated from the ruptured muscle. The cavity was irrigated with saline. No bleeding point was demonstrated and the wound was closed with interrupted cotton sutures in layers and without a drain. (Fig. 3.)

Following an uneventful postoperative course the patient was discharged on the third postoperative day.

Final diagnoses: (1) hematoma of the abdominal wall (superior half of right rectus abdominis muscle); (2) status post-typhoid fever.

Case IV. This sixty-two year old white woman was admitted to the medical service on February 21, 1950, because of tightness in her chest and coughing of several days' duration. The initial

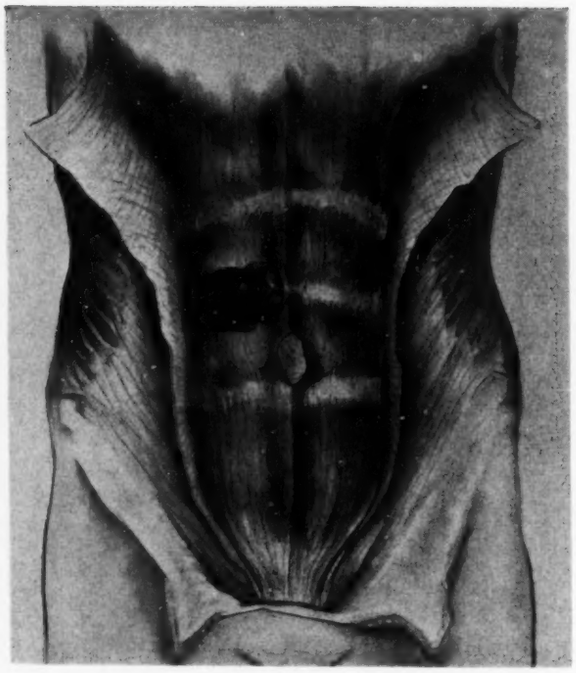

FIG. 3. Location and relative size of hematoma of abdominal wall in Case III.

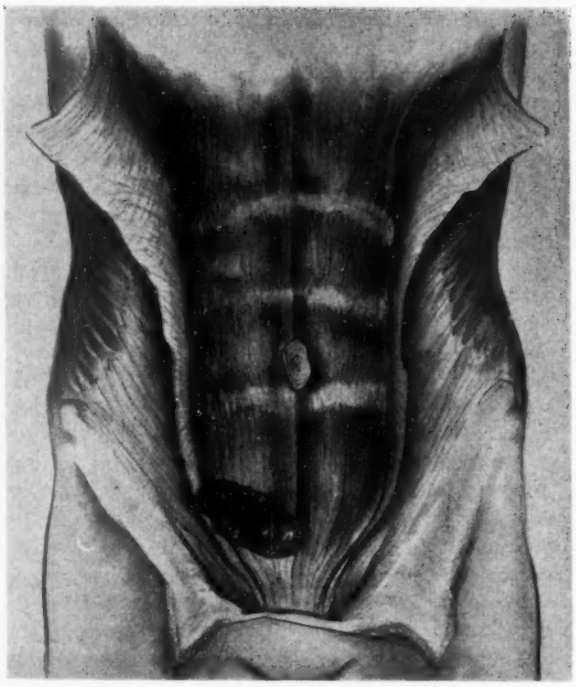

FIG. 4. Location and relative size of hematoma of abdominal wall in Case IV.

diagnosis was acute bronchitis, which subsided with medical treatment.

The patient denied any history of typhoid fever. Some years previously she had had an operation on her internal reproductive organs.

On physical examination the patient had pulmonary findings compatible with acute bronchitis. Her temperature was 101°F. and her pulse was 100. On the day of admission slight tenderness was noticed just above the right inguinal ligament. The remainder of the examination was essentially negative.

Laboratory data were as follows: The initial white count was 4,900. The urinalysis and erythrocyte count were normal.

On February 24th, three days after admission, the patient complained of pain just above the right inguinal ligament. In addition, her coughing continued but appeared to be less frequent and intense. On March 1st tenderness was quite marked just above the right inguinal ligament and a mass was felt for the first time. In addition, the total white count had risen to 18,850. A barium enema x-ray examination on February 27th showed no abnormality. On February 26th deep induration was noted over the right inguinal canal and Dr. Wells Teachnor was asked to see the patient in surgical consultation.

The preoperative diagnosis was (?) incarcerated, right, indirect, inguinal hernia.

Operation was performed on March 2, 1950, under pontocaine spinal anesthesia. Through a right lower rectus incision a large blood clot was found in the right rectus abdominis muscle. The muscle itself appeared to be ruptured, and the muscle ends were separated for about 5 cm. by a large blood clot which was evacuated. The inferior epigastric artery which appeared to be responsible for the hematoma was ligated at the inferior limit of the clot. On opening the peritoneum, no free blood or hernias were encountered. The wound was closed in layers without a drain. (Fig. 4.)

The postoperative course was excellent with no complications, and the patient was discharged on March 24th.

Final diagnoses: (1) hematoma of the abdominal wall (inferior half of right rectus abdominis muscle); (2) acute bronchitis.

CASE V. Three weeks prior to admission this forty-one year old white woman noticed dyspnea, orthopnea and a non-productive cough which continued up until the present admission. On the day prior to admission she had a bout of rather severe paroxysmal coughing, and since that time had severe abdominal pain, mainly in the left upper quadrant, which came after a sensation that "something had given way." She became nauseated, vomited and became quite thirsty.

Her last menstrual period was approximately one week prior to admission and had been normal. At the age of forty appendectomy had been performed through a right lower quadrant incision. She had been treated for hypertensive cardiovascular dis-

FIG. 5. Location on roentgenogram of hematoma of abdominal wall in Case v. Outline of the hematoma indicated by the arrows.

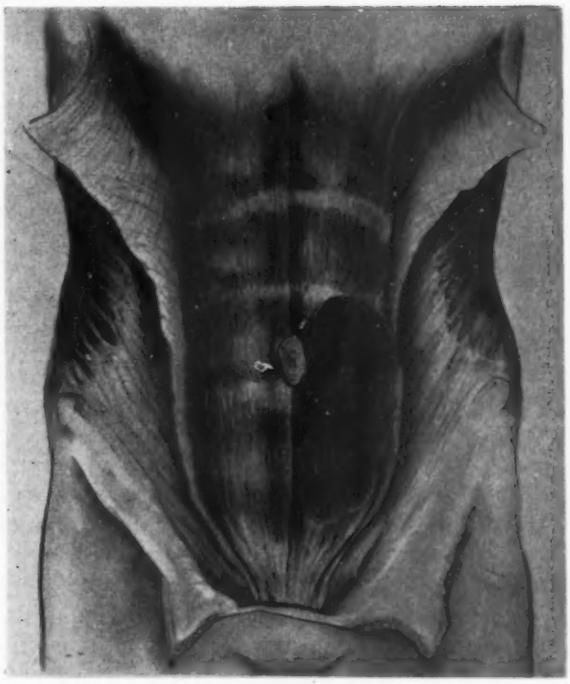

FIG. 6. Location and relative size of hematoma of abdominal wall in Case v.

ease and chronic pyelonephritis for four years, and had been hospitalized a number of times. She denied a history of typhoid fever.

On physical examination the patient seemed to be an acutely ill, well nourished and fairly well developed white woman of approximately her stated age. She was oriented and generally cooperative. Her temperature was 97.8°F., pulse 100, respirations 24 and blood pressure 196/110. Lungs were clear to percussion and auscultation except for an occasional rough sound in the tracheal region and in the region of the large bronchi. There was a sinus rhythm of her heart with a grade II systolic murmur at the apex. The abdomen was large and protuberant, and exhibited definite muscle guarding over the left rectus abdominis muscle. There appeared to be a large mass in the lower quadrant of the abdomen. Tenderness was quite marked when the left side of the abdomen was palpated. Bowel sounds were present. The pelvic examination seemed within normal limits although it was difficult to palpate the adnexa in the region of the left tube and ovary because of the severe pain and muscle guarding which were present during the examination. The extremities were normal with the exception of 1 plus ankle edema.

Laboratory data were as follows: The total erythrocyte count was 3,670,000; hemoglobin 12.4 gm. per cent; and total leukocyte count 8,000 with an essentially normal differential count. Urinalysis was within normal limits with the exception of 100 mg. per cent albumin, granular casts and hyaline casts.

Emergency barium enema study showed no abnormality except for some fixation and improper filling in the region of the right colon. On an antero-posterior abdominal film a soft tissue shadow about 10 cm. in diameter was seen to be present on the left side of the abdomen. (Fig. 5.)

Operation was scheduled as an emergency procedure and indicated supportive measures were carried out.

The preoperative diagnosis was (?) twisted ovarian cyst with hemorrhage.

At operation on February 13, 1952, under pontocaine spinal anesthesia, a left paramedian incision was made. As the anterior rectus sheath was opened, a very large hemorrhagic clot was found in the region of the rectus abdominis muscle. A complete placenta basin full of blood was removed from the region of the muscle. It was apparent that the operator was dealing with a massive hemorrhage probably due to rupture of the inferior epigastric artery. The hemorrhage was rapidly controlled. During this procedure the patient became cyanotic, the blood pressure fell to zero and cardiac arrest occurred. Artificial respiration with the aid of an endotracheal tube and cardiac massage for one hour, electric shock, intracardiac adrenalin® and procaine applied to the surface of the heart failed to make the patient respond. (Fig. 6.)

Noted at autopsy was the large cavity in the left

rectus abdominis muscle in which the hematoma had developed, along with the findings of hypertensive cardiovascular disease with congestive failure.

Final diagnoses: (1) massive hematoma of the abdominal wall (inferior and superior portions of left rectus abdominis muscle); (2) cardiac arrest; (3) hypertensive cardiovascular disease with congestive failure.

SUMMARY AND CONCLUSIONS

1. Although hematoma of the abdominal wall is an infrequently seen clinical entity, a correct preoperative diagnosis can be made if the surgeon is "hematoma conscious."

2. Correct preoperative diagnosis makes possible a decrease in the number of diagnostic tests, allows for immediate correct therapy and lessens the duration of the patient's hospital stay.

3. The anatomy of the rectus abdominis muscles, of the epigastric vessels and of the anterior abdominal wall has been reviewed.

4. Predisposing and exciting etiologic factors have been discussed.

5. The clinical picture has been illustrated with data of five cases reported in this paper and seventy-nine cases presented in the literature during the period from 1939 to 1955, inclusive.

6. Preoperatively, hematoma of the abdominal wall may simulate any of the myriad intra-abdominal and abdominal wall conditions which may be responsible for an acute surgical condition of the abdomen.

7. Treatment may be operative or non-operative. Usually it is surgical incision and evacuation of the hematoma.

8. The mortality rate is low and may be more a reflection of the predisposing and exciting factors than of the hematoma itself.

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Pancreatic Injuries Due to Non-penetrating Abdominal Trauma

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PANCREATIC injuries due to trauma are of three etiologic types: operative, penetrating and non-penetrating.[7] Surgical trauma is by far the most common of these.[7] Penetrating wounds of the pancreas, not commonly encountered in civilian hospitals, are best considered under the general problem of abdominal penetrations.

Blunt injuries of this organ occur more frequently[5,25] than generally recognized (Table 1), and for this reason deserve further emphasis.

Mechanism. The pancreas, while lying in the dorsal region of the abdomen, has the disadvantage of fixation over the first lumbar vertebra. This anatomic situation lends itself readily to the classic contrecoup type of injury following blows to the upper abdomen. In the day of super horsepower automobiles the steering wheel injury is often responsible for this condition. Other blows, as from a policeman's billy, bicycle handlebars and baseball bats, are causative. Accident victims that are thrown from, run over, or crushed by automobiles are particularly prone to injury of the pancreas. It is significant that even trivial blows may be followed months later by symptoms referable to the pancreas.[3] The widespread use of alcohol, with its attendant stimulation of the pancreas,[12] may predispose the victim's injured pancreas to inflammation.

TABLE 1
FREQUENCY OF BLUNT TRAUMA TO PANCREAS

Hospital	Years	Acute Pancreatitis	Blunt Trauma	Pancreas Alone
San Francisco Hospital	1942–1950	171	9	5
St. Luke's Hospital, Bethlehem, Pa.	1935–1951	.....	4	3
Los Angeles County Hospital	1939–1951	1,328	18	14
Charity Hospital of Louisiana	1944–1954	109	6	..
Ohio State Hospital	1948–1954	.....	5	..
Harlem Hospital	1936–1946	75	4	..
Georgetown University Hospital	1946–1951	135	4	3
Louisville Veterans Administration Hospital	1946–1954	155	4	4

Pathology. Injuries to the pancreas can be conveniently divided into three types as follows: (1) simple contusion with edema, (2) hematoma or hemorrhage, and (3) varying degrees of lacerations to complete transection of the pancreas.[3]

The typical inflammatory response to injury occurs, and in addition, acinar leakage of pancreatic ferments with activation may result. The exact mechanism of activation is not clear (various theories relate it to necrotic tissue, bile, ischemia or bacteria). The enzyme lipase speeds the breakdown of fat to glycerin and fatty acids. Calcium then combines with the fatty acids to produce typical findings of fat necrosis, which is a process of saponification. Activated trypsin may break down cellular protein, and in so doing open up damaged blood vessels, leading to further delayed hemorrhage. Amylase is apparently absorbed from the damaged organ, and a resultant rise in serum amylase occurs. If the damage is sufficient, an outpouring of these secretions and an attempt by the body to localize this process will result. This gives rise to the characteristic pseudocyst which may gradually enlarge if there is continued leakage of secretions particularly when there is obstruction or damage to the proximal duct system. Likewise, enzymatic breakdown of hematoma may result in pseudocyst formation. If the inflammation subsides spontaneously, fibrosis (or calcification) of the pancreas may result which may be

severe enough to produce diabetes or digestive disturbances.

Clinical Picture. When the pancreas alone is injured, the symptoms and physical findings vary depending on the severity. There is often a delay of twenty-four to seventy-two hours in onset of the complaints.[2,7] Pain is always present and may be mild to excruciating. It is usually upper abdominal (left, right or epigastric), not necessarily band-like, and frequently is diffuse. Radiation to back and shoulders occurs at times. Nausea and vomiting may be pronounced. Shock, contrary to most books, is rare[2] except with complete transection where major vessels are severed. Physical findings are tenderness with guarding or rigidity, and rebound of the upper abdomen, although occasionally these signs may be more diffuse. A low grade temperature elevation is most often present.

In approximately 50 per cent of cases, there are important associated intra- and extra-abdominal injuries which may mask the pancreatic injury. Head injuries, rib, long bone and spinal fractures are common, and too much stress cannot be placed upon diligent observations for the development of abdominal symptoms in these unfortunate accident victims. Lacerations of the liver, spleen and kidney are the most frequently[3] associated intra-abdominal injuries. The signs and symptoms following injuries to these solid viscera or to the hollow viscera may overshadow pancreatic trauma. A not too common, but grave, associated injury is retroperitoneal laceration of the duodenum.[3,8] Occasionally, this suspected injury may be diagnosed clinically by the presence of air along the psoas muscle on x-ray of the abdomen or by the instillation of lipiodol via a Levin tube with x-ray demonstration of leakage from the duodenum.

If there is doubt concerning the diagnosis (that is, suspicion of other injuries), an extremely worth while procedure is the use of paracentesis to obtain peritoneal fluid.[2,8] This may be done in the right lower quadrant with the patient in the right lateral position (or even attempted in all four quadrants as suggested by Prigot[8]). The fluid in typical pancreatic injuries is thin and blood-tinged, and amylase determinations (on as little as 0.2 cc.) are reported anywhere from 5,000 to 50,000 Somogyi units. The presence of bile or intestinal contents in the fluid can easily be ascertained. This procedure has been employed in a number of instances at the Louisville Veterans Administration Hospital, and so far as we know there has been no morbidity or mortality.

Laboratory. Elevation of the serum amylase is a definite aid in diagnosis. This test is quickly and easily run, and most laboratories report results in Somogyi units, the normal being from 60 to 200 (varies with hospitals). Characteristically, the amylase level may fall to normal within forty-eight hours. It is by no means infallible, as high amylase may result from upper intestinal perforation or following use of morphine SO_4 with production of spasm of the sphincter of Oddi. A rising amylase is, of course, more diagnostic than an isolated elevated reading.

The white blood count most often is elevated to 10,000 to 15,000.

The red blood count and hemoglobin are within normal limits in the early phase unless there has been massive hemorrhage, but fall in the red cell mass as well as plasma volume can be anticipated.

As fat necrosis is not often extensive in comparison to non-traumatic pancreatitis, the level of serum calcium is usually normal.

Scout x-ray films of the abdomen often reveal some degree of localized ileus, the so-called "sentinel loop."

Management. The following are general principles of management applied to injuries to the pancreas alone. These should not be construed as a conservative regimen; rather they are the basis of active treatment in both operative and non-operative cases.

1. Reduction of pancreatic secretion, accomplished by: (*a*) Absolute starvation to diminish gastric secretory activity and hence humoral (secretin) stimulation of the pancreas. (*b*) Nasogastric indwelling tube with removal of stomach contents, which has dual purpose of decreasing pancreatic secretions and relieving distressing ileus. (*c*) Parasympatholytic drugs which block the neurogenic (vagal) control of pancreatic function. Atropine is effective although pro-banthīne® is the drug of choice. Pro-banthīne is given in doses of 50 mg. intravenously, four times a day, until tablets can be substituted. These drugs, in addition, reduce gastric hydrochloric acid formation.

2. Reduction of pain by the use of: (*a*) Demerol,® 100 mg. every three to four hours. Morphine sulfate is contraindicated as it tends

to produce spasm of the sphincter of Oddi. (*b*) Continuous epidural analgesia.[6] In the past, splanchnic or lumbar paravertebral blocks were used to interrupt the visceral afferent fibers from the pancreas. Although these were often effective, epidural block has recently proved to be more efficient with the added ease of intermittent injections. A small catheter is passed to the level of the eighth thoracic vertebra and procaine is injected once to twice per twenty-four hours.

3. Prevention of abscesses, or control of secondary infection, with the broad-spectrum antibiotics.

4. Repeated intravenous injections of serum albumin have been shown to have a decidedly beneficial effect on non-traumatic pancreatitis,[9] and probably traumatic pancreatitis as well. This has been attributed to its known antitryptic effect, although present work suggests that this is due simply to replacement of depleted body proteins.

5. Finally, rigid control of the body electrolytes and fluid requirements must be exercised, as well as replacement of any defects in this system. Under this heading would come prophylaxis or treatment of hypocalcemia.

Operative Principles. Extreme conservatism[15,16] as regards surgery in all types of pancreatitis has been practiced in the past, and this is true today in a number of clinics. This reluctance to operate stems from the high mortality reported previously. On the other hand, there are clinics where immediate surgery with drainage is the recommended procedure for all cases of traumatic pancreatitis.[8,25] However, the majority of writers seem to favor non-operative management of the mild cases and reserve operative intervention for those cases which fail to improve or worsen after the institution of the foregoing plan of therapy.[3,5,15] In this connection, I would like to quote from Dr. Joseph Hamilton's discussion of a paper on this subject at the meeting of The American Association for the Surgery of Trauma in 1951.[4] He stated, "It would seem that the great amount of highly active juice, as well as bits of digested pancreas that drain from some of these patients are better outside than inside the abdomen."

In undertaking exploration of this type of injury it is mandatory that the duodenum be mobilized and inspected in its retroperitoneal areas for perforation, even in the absence of the typical green-black peritoneal discoloration.[3] In the presence of contusion with edema or hematoma, simple drainage of the involved areas is indicated. Commonly, drains are placed behind the mobilized duodenum to the head, through the gastrocolic ligament, to the body and tail, and via the lesser omental bursae to the body. The use of a sump type of drain will obviate difficulty with erosion of the skin.

When extensive or complete lacerations are present, careful attention to the injured ducts is required. If the proximal duct is transected, repair may be necessary. Distal duct injuries can be controlled by ligation. Devitalized portions of the gland should be excised. The cut ends, in cases of complete transection, should be closed with mattress sutures. Adequate drainage once again in *all* cases should be exercised.

If there are serious associated intra-abdominal injuries (such as to the liver, spleen or hollow viscera) with warning symptoms and signs, certainly exploration and management according to established practices is carried out. Pancreatic injuries discovered at such times are handled as noted heretofore.

Mortality. No large series have been reported, but it was possible to review case reports of forty-seven instances of blunt trauma to the pancreas alone over the past ten years.[3,7,8,10,15,18,19,22,23,25,26,29,30] There were three deaths among these cases, a mortality rate of 6 per cent. There is no question but that the mortality in traumatic pancreatitis with associated injuries is higher and reflects the seriousness of injuries to the spleen, liver, hollow viscera and so forth.[3]

Sequelae and Treatment. Fistulas are common sequelae, usually occurring in the operative cases. These tend to close spontaneously,[26] provided there is no obstruction (damage) to the proximal duct system. Management of fistulas revolves around protection of adjacent skin and control of electrolytes. Persistent fistulas may require surgery[7] with excision of a portion of the pancreas or implantation of the fistula.

Pseudocysts may develop in either operative or non-operative cases. Rarely the cysts subside spontaneously, although the large majority require surgical drainage. It is preferable to withhold treatment until the cysts enlarge and produce symptoms. Treatment of choice

is some type of internal drainage such as cystogastrostomy or cystojejunostomy. Marsupialization may be used in poor risk cases, but persistent fistulas may result. Occasionally cysts may be excised if location is favorable.

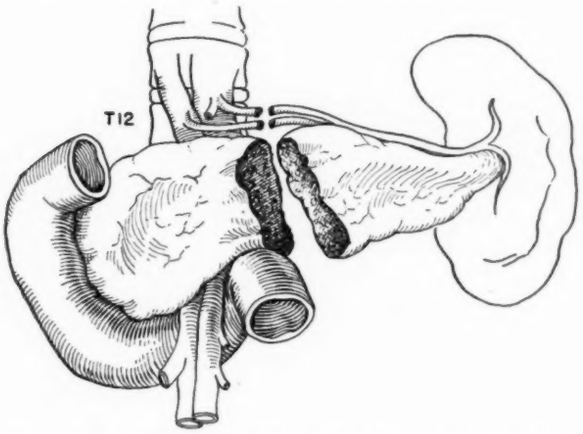

FIG. 1. Case I. Blunt abdominal injury with complete division of the pancreas.

Impermanent or permanent diabetes can be controlled by usual dietary regulation with insulin therapy.

Digestive disturbances, such as steatorrhea due to pancreatic insufficiency, are a problem; however, replacement therapy with pancreatin (8 gm. three times a day) is of value.[7,13]

Chronic recurrent pancreatitis may follow, and this is perplexing and difficult as evidenced by the multiplicity of operative procedures proposed for its solution. Time will not permit discussion of procedures designed for this distressing complication.

CASE REPORTS

CASE I. P. C., a twenty-eight year old white man, was admitted on January 6, 1954, with a history of crushing injury (railroad cars). The patient exhibited profound shock and his abdomen was rigid. On admission the white blood count was 22,900. At operation 3,500 cc. of blood were present in the peritoneal cavity. There was complete laceration of the distal pancreas and splenic vessels. (Fig. 1.) Distal pancreatectomy was performed with splenectomy and drainage. Moderate drainage from the pancreas occurred with no excoriation. On the fifth postoperative day therapy was begun with anticoagulants as a prophylactic measure in view of a rising platelet count. Death resulted from massive hemorrhage on the eighth postoperative day before emergency measures could be instituted. Postmortem examination showed no definite site of hemorrhage.

Comment. In cases of complete transection such as this one the clinical picture establishes the need for immediate laparotomy. Although this type of injury has carried a high mortality in the past, with present-day management

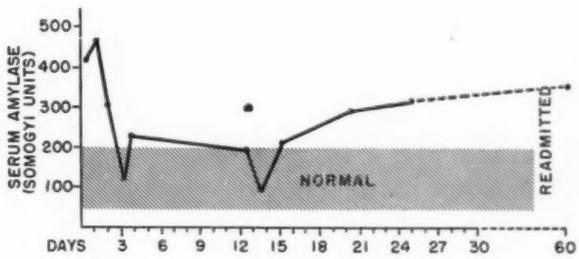

FIG. 2. Case IV. Amylase values in case of severe trauma (blunt) to pancreas.

they should have an equally good chance for survival.

CASE II. W. H., a sixty-three year old white man, was admitted on January 8, 1953, with a history of having been in an automobile accident. The patient apparently was inebriated at the time of the accident. Severe pain and upper abdominal tenderness and rigidity were present. Fracture of the fifth and sixth ribs on the right was noted, with pneumothorax as well as suggestive subluxation of the second lumbar on the third lumbar vertebra. The white blood count was 13,800. Serum amylase was 520 Somogyi units. Paracentesis yielded bloody fluid with amylase of 5,139 Somogyi units. At exploration there was dark, thin bloody fluid with diffuse fat necrosis. The pancreas was edematous with hematoma. The gallbladder was found to be tense and non-compressible. Operation was cholecystostomy with drainage of the pancreas. Psychosis developed on the sixth postoperative day. A fistula drained for eight weeks but was finally controlled by catheter suction. On follow-up examination six months later, the patient was asymptomatic.

Comment. This patient was explored as he was not improving under treatment, and the peritoneal tap suggested significant intraperitoneal hemorrhage. The development of psychosis has been reported by others.[23]

CASE III. R. M., a fifty-eight year old white man, was admitted on November 10, 1952, with a history of crushing injury (truck and loading platform). A large 5 by 7 cm. movable tender mass was present in the left hypochondrium. Vital signs were stable. The white blood count was 19,000. Amylase was 176 Somogyi units. At exploration there was massive hematoma of the pancreas as well as the base of mesentery, mesocolon and duo-

denum. Suggestive fat necrosis was noted. Simple drainage was instituted. No fistulas developed postoperatively, but the mass remained, apparently as a pseudocyst. The patient was discharged, only to return two weeks later with an acute con-

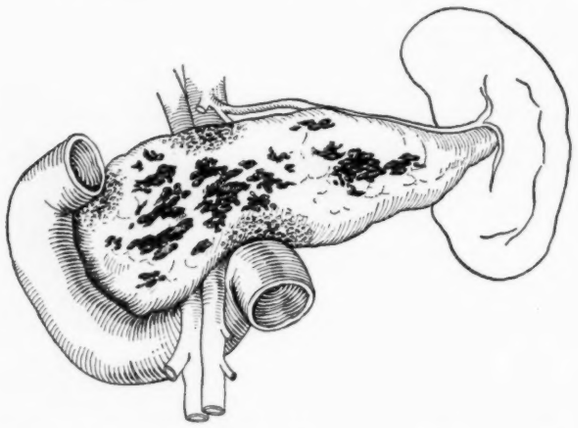

FIG. 3. Case IV. Edema and hemorrhage of pancreas at time of exploration following blunt trauma.

dition of the abdomen and disappearance of the mass. Serum amylase at this admission was 921 Somogyi units. At operation the large pseudocyst of the pancreas had ruptured into free peritoneal space. The cyst was drained. Amylase on the cystic contents was 40,000 Somogyi units. The fistula closed in several months. Ten months later, at the time of ventral hernia repair, there was only residual nodularity of the head of the pancreas. The patient is now asymptomatic.

Comment. This spontaneous rupture of the pseudocyst is quite unusual, although Koucky and co-workers reported six cases.[28] The tendency toward spontaneous closure of the fistula is illustrated by this patient.

CASE IV. K. B., a twenty-eight year old Negro man, was admitted on May 18, 1951. His abdomen had been stomped on by assailants forty-eight hours previously. The white blood count was 8,700, with 36 stab forms. Amylase was 410 Somogyi units. (Fig. 2.) Vital signs were stable. Abdominal findings were diffuse. At exploration there was dark brown, thin fluid, with fat necrosis of the omentum. (Fig. 3.) The pancreas was markedly edematous and rubbery. The pancreas was drained. Four weeks postoperatively the patient was reoperated upon, with release of obstructing, encircling scars of the distal portion of the stomach. The pancreas at this time showed marked hemorrhage and necrosis. A fistula was a difficult problem but it closed in three months. The patient developed toxic psychosis due to atropine poisoning. Four months after discharge he was asymptomatic. Shortly thereafter he was sent to prison for chicken stealing. Reports from the patient's mother indicate he is asymptomatic but she states, "Doctor, he just don't act right anymore."

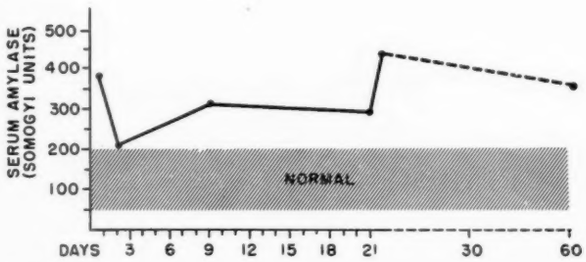

FIG. 4. Case V. Persistent elevation of serum amylase in patient with blunt pancreatic injury.

Comment. We had an unusual opportunity to see the injured pancreas on two occasions in this case, and it was impressive to note the progression of the edematous pancreas to that of hemorrhage with necrosis. This patient was extremely sick and he presented many problems in management of the severe injury to the pancreas. The psychosis following long-continued atropine cleared rapidly following its cessation.

CASE V. D. J., a thirty-three year old white man, was admitted on July 12, 1952, with a history of being thrown from a truck thirty hours prior to admission. Apparently he was inebriated at the time of the accident. There was diffuse abdominal pain, along with tenderness and spasm. Fracture of the ninth and eleventh ribs on the left side, and also compression of the eleventh thoracic vertebra were noted. Vital signs were stable. Amylase was 344 Somogyi units. (Fig. 4.) The white blood count was 14,000. Non-operative management was instituted, with improvement. On the eighth day the patient became psychotic; this condition was attributed to massive atropine therapy. Nineteen days later recurrence of symptoms developed, which was quickly controlled by banthīne® therapy. He returned one month after discharge with symptoms of pancreatitis, again controlled by banthīne. Follow-up two years later revealed a history of occasional epigastric pains, nausea and vomiting. Work-up at that time was negative.

Comment. The clinical picture and the response to non-operative management established the diagnosis in this case, and we were able to continue without surgery; at times, however, we were on the verge of operating upon this patient. This patient demonstrated again a toxic psychosis from atropine. Banthīne

was substituted and it was effective in controlling symptoms for several months. We suspect this patient will continue to have difficulty, as he will abuse his pancreas with bouts of heavy drinking.

SUMMARY

1. Blunt trauma to the pancreas occurs more often than is generally recognized.

2. This injury is frequently associated with other intra- and extra-abdominal injuries.

3. A plan of non-operative treatment of all cases has been outlined.

4. Operative intervention, according to principles listed, is reserved for the more serious cases and for those cases in which there are grave associated intra-abdominal injuries.

5. The mortality rate in pancreatic injuries alone, including both operative and non-operatives cases, is around 6 per cent, according to the recent literature.

6. Five cases, personally observed, have been reported.

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Injuries to the Appendix Secondary to Blunt Trauma

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THE purpose of this paper is threefold: first, to present a case of traumatic appendectomy secondary to non-penetrating abdominal trauma; second, to call attention again to the relationship of trauma to appendicitis; and finally, to present a second case illustrating the difficulty in diagnosing appendicitis, traumatic or otherwise, in the presence of acute trauma.

We have always questioned, as most surgeons undoubtedly do, that trauma causes acute appendicitis. We have, in fact, believed that the ruling of the Works Progress Administration Commission[1] in Washington in the early thirties was correct in holding that ". . . there is no such entity as traumatic appendicitis."

However, the senior author, on putting his hand into an abdomen, found a detached appendix and its attached mesentery. This finding proves that if trauma can cause an amputation of the appendix it can also initiate inflammation of that viscus. The case in point follows:

M. O., a thirty-nine year old woman, was admitted to the accident ward of Douglas County Hospital at 11:45 P.M., October 21, 1951, following an automobile accident. She had been one of three persons riding in the front seat of a car when it was struck by another vehicle.

On admission the patient complained of pain in the left knee and right elbow. She had not been unconscious. Careful examination of the abdomen revealed only a thick fat pad compatible with her weight of 150 pounds. She had no abdominal complaints. A roentgenogram of her left knee revealed a comminuted fracture of the tibial plateau.

Three hours later she complained of some abdominal pain. The abdomen was then rigid and there was a bruised area in the right lower quadrant. An x-ray film of the chest and a flat plate of the abdomen were of no definite diagnostic help.

With a preoperative diagnosis of trauma to the abdominal wall and rupture of the liver, the abdomen was explored through a right rectus incision. A contusion of the right mid-abdominal wall was found, with hemorrhage into the subcutaneous fat. The right rectus muscle had been torn across and the peritoneal cavity contained a great deal of blood. When the examining hand was inserted into the abdomen, the distal three-fourths of the appendix with its mesentery was found to be floating free in blood and fecal material. A 12 inch laceration extended along the mesentery of the terminal ileum to its base, and approximately 3 feet of the ileum were dark red from loss of blood supply.

The 1.5 cm. stump of the appendix was removed and bleeding from the mesentery was controlled. Approximately 3 feet of the terminal ileum were resected and a lateral anastomosis was performed. The abdomen was drained with a Penrose drain brought out through a stab wound to the right, and the abdomen was closed in layers.

Pathologic report of the tissues removed at operation was as follows:

"Gross—Distal 6 cm. of appendix with attached fat. The mesenteriolum is firm, indurated and hemorrhagic. The proximal end of the lumen is irregular, rough and torn and there is hemorrhage into the wall. The surface of the appendix is hyperemic and slightly granular. On section the lumen contains mucoid and fecal material. 71 cm. of resected ileum and mesentery with red wall and dull rough peritoneal surface.

"Histology—Appendix with hemorrhage into proximal portion along with some acute inflammatory reaction and necrosis. Hemorrhage into mesenteriolum and acute inflammation.

"Ileum—Infarction with early acute peritonitis.

"Diagnosis—Traumatized appendix with acute peri-appendicitis; early infarction of resected distal ileum; laceration and hemorrhage of small bowel mesentery."

Convalescence was complicated by a spiking type of fever and diarrhea. A pelvic abscess developed which required drainage through the vagina. Following this procedure recovery was complete.

Because the patient had been asymptomatic prior to the accident and because five and a half hours had elapsed from the time of injury until surgery, thus accounting for the inflammatory reaction (periappendicitis), we believe that this was an instance of traumatic appendectomy of a

previously normal appendix. Apparently the injury only partially amputated the mesentery of the appendix and enough of its blood supply remained to permit development of some inflammatory reaction on its peritoneal surface.

The second case is that of A. W., a sixty-six year old woman, who was admitted to St. Catherine's Hospital on March 29, 1949, with the following history:

She had felt well until March 29th, when she fell down while going through a revolving door. After the fall she had pain in the right hip and was unable to walk.

On admission she had no abdominal complaints although the abdomen was noted as being greatly distended on physical examination. Her blood pressure was 130/80 and her pulse 100. X-ray films revealed a subcapsular fracture of the neck of the right femur; the distal fragment was displaced upward and externally rotated.

The next morning, about twelve hours later, she complained of abdominal discomfort and gaseous distention. A blood examination then revealed an elevated white count with a shift to the left. The hemoglobin was 87 per cent; erythrocytes, 5.02 million; leukocytes, 12,300 with 66 per cent segmented and 12 staff forms; 1 basophil, and 21 per cent lymphocytes. The urine was normal.

On the fourth hospital day flat films of the abdomen revealed gaseous distention of both small bowel and colon, more suggestive of an adynamic ileus than of mechanical obstruction. It was believed that abdominal distention was due to retroperitoneal hemorrhage secondary to fracture. The patient seemed to improve for several days although abdominal distention was always present. On the eighth day her white cell count was 34,000, with 21 per cent staff forms. She appeared gravely ill with marked sepsis and it seemed best to continue treating her expectantly. She died on April 13, 1949, fourteen days after admission.

Autopsy indicated that death was due to acute peritonitis following perforation of the appendix. Pertinent findings were as follows: "On opening the abdomen there was an escape of foul smelling gas. The bowels were all distended and flecked with fibrin and the loops were bound together by masses of fibrin. The right side of the abdomen appeared to be more involved with the acute peritonitis, although the process had extended throughout the entire abdomen. In the right lower quadrant there was a large thick mass, apparently a large abscess, partially walled off from the general peritoneal cavity. On breaking into this abscess a large appendix, about 10 cm. in length and up to 2 cm. in greatest width, was seen. It was flecked with fibrin, hyperemic and there were a number of dilated blood vessels in the area. At the base of the appendix there was a perforation 5 mm. in greatest diameter, from which issued a large amount of semi-solid jelly-like material. What appeared to be bowel contents could be freely expressed. There was a large amount of purulent material under each leaf of the diaphragm."

COMMENT

Although traumatic rupture of the appendix has been recognized as an entity for years, we can find in the literature no instance of traumatic amputation of a normal appendix.

Burgess[2] reported an amputation of the tip of the appendix at the site of an old adhesion after the use of a pneumatic drill which rested in the right lower quadrant of the patient's abdomen.

Mannini[3] cited the case of a thirty-six year old laborer who was struck on the abdomen by a large stone. Laparotomy for acute appendicitis performed the following day showed a 7 cm. appendix, partly gangrenous and perforated at the base, floating free in the abdomen. Acute general peritonitis caused death.

Neuman[4] reported the case of a twenty year old man in whom pain developed in the right iliac fossa shortly after he lifted a heavy weight. Nine days later he was hospitalized, and at operation only a thickened stump of the appendix (1.5 cm. long) was found. A concretion the size of a hazelnut lay free in the abdominal cavity.

Kelly[5] reported a similar case. A forty-four year old man felt a sharp pain in the right side of his abdomen after strenuous lifting. Operation, five days later, disclosed that the appendix had sloughed off from its base. It contained a concretion in the tip.

In our first case it is important to note that on admission the patient did not have abdominal complaints. This is indicated by the fact that neither the intern nor the attending physician ordered a blood count. It was not until three and a half hours later that she complained of abdominal discomfort and examination disclosed rigidity. The physician must remember that lack of pain is not unusual following severe trauma and he must watch such patients closely lest delay be even longer than in our case.

Our cases support Stewart's[6] statement that "It is notable that in certain instances of blunt trauma to the abdomen, serious visceral lesions may result with but little early indication of their presence. In some cases the small intestine

may be completely transected, with little or no hemorrhage, so that the individual appears to be free of trouble until a rapidly spreading peritoneal contamination results."

The exact mechanism of injury in our case is difficult to state. The abdominal wall was well padded and the cecum was in normal position. Because the cecum was fixed and the rent in the mesentery involved the terminal ileum, it is likely that a tangential force could have caused this injury by striking the appendix and its mesentery and then the mesentery of the ileum, and compressing them against the retroperitoneal tissue or spine and tearing them. Rupture of the right rectus muscle was probably caused by the same blow but could have resulted from violent contraction of the muscle.

Our second case supports Boyce's[1] statement that the frequent delay in diagnosis of traumatic appendicitis is due not to lack of symptoms but to confusion caused by the history of trauma. Certainly, in reviewing this case, it would seem difficult not to entertain the diagnosis of appendicitis in a patient who had been well until mild abdominal symptoms developed after injury, with elevated white blood count, distention and ileus, and subsequent death of the patient. Of course the presence of a fractured hip directed our attention away from the abdomen, as it did in Boyce's patient who died of generalized peritonitis secondary to rupture of the appendix fourteen days after injury.

One can only say that a diagnosis cannot be made unless it is considered. An obvious injury should not be allowed to obscure our thinking so that another injury or disease is overlooked.

CONCLUSION

1. As far as we can ascertain we have presented the first reported traumatic amputation of a normal appendix following non-penetrating abdominal trauma.

2. A case of traumatic rupture of the appendix also has been presented.

3. The lack of pain for a time following severe abdominal trauma must be kept in mind so that intra-abdominal injury will not be overlooked and exploration will not be too long delayed.

4. One must not be misled by trauma to other parts of the body and therefore fail to suspect and overlook signs of intra-abdominal trouble.

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Traumatic Rupture of the Spleen, with Special Reference to Delayed Splenic Rupture

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THE mortality rate of acute traumatic rupture of the spleen has been reduced in recent years because of the greater availability of blood. Yet the mortality rate of delayed splenic rupture has continued to be high, largely because of inability to diagnose the situation correctly and to perform splenectomy prior to death from exsanguinating secondary hemorrhage.

Incidence of Splenic Rupture. The problem of traumatic rupture of the spleen is of increasing importance because of the increasing number of injuries resulting from travel at high speed. This is borne out by our experience at the St. Francis Hospital: During the period from 1940 to 1947 there were but three cases of splenic rupture, whereas from 1947 to 1955 there were ten such cases. During the latter period a similar review of splenic injuries was carried out at the 1100th U.S.A.F. Hospital and Walter Reed Army Hospital, and eleven additional cases of splenic injury were recorded, a total of twenty-four cases in all.

At the St. Francis Hospital the frequency of splenic rupture when related to traumatic admissions was 0.15 per cent of all such admissions. This incidence is comparable to the 0.11 per cent reported by Roettig and his associates[1] from the University Hospitals, Columbus, Ohio, and by Wright and Prigot,[2] who reported a 0.15 per cent incidence at the Harlem Hospital, New York, New York.

In our experience splenic rupture occurs about equally in men and women (seven and six, respectively). As would be expected, however, in the military hospitals only a small proportion (two of eleven cases) occurred in women.

As in most series of splenic rupture, over 80 per cent of our cases were in the age group below forty; it is this group which is most often exposed to the possibility of severe trauma.

Incidence of Delayed Splenic Rupture. The incidence of delayed splenic rupture is of special importance since this is the group in which errors in diagnosis are most likely to occur. Eight of our twenty-four cases of splenic rupture (33.3 per cent) were of the delayed type. This is a higher incidence of delayed rupture than has been reported by other observors (Table I); but since our group of cases is small, it does not necessarily indicate an increasing frequency of this type of splenic injury.

We can say, however, that since more than one in five splenic ruptures are of the delayed type, we must be alert to the possible existence of delayed rupture where circumstances suggest its possibility.

Etiology of Splenic Rupture. We have already suggested that high speed travel is implicated in the increasing incidence of splenic rupture. (Table II.)

Pathology of Splenic Rupture. Rousselot's[6] classification of splenic trauma into the various pathologic groups (hematoma, tear, multiple lacerations and splenic avulsion) makes it

TABLE I
RELATIVE INCIDENCE OF ACUTE AND DELAYED SPLENIC RUPTURE

Author	No. of Cases	Percentage of Delayed Rupture
Byrne[3]	101	26.7
Zabinski and Harkins[4]	111	14.5
Dobson[5]	22	22.7
Bollinger and Fowler	24	33.3
Totals	258	21.5

evident that those cases of splenic avulsion or multiple lacerations will, due to rapid blood loss, usually fall into the acute splenic rupture group clinically and will be so diagnosed with little difficulty and then promptly treated. Those cases in which there is a hematoma which subsequently bursts, or a small tear in the splenic capsule with only minimal bleeding, will most commonly present the clinical picture of delayed splenic rupture. Since the symptoms and signs of delayed splenic rupture are not always characteristic and because the picture may often be confused by associated injury, we shall briefly mention those which in our experience have been most useful in arriving at a correct diagnosis.

TABLE II
ETIOLOGY OF SPLENIC RUPTURE IN AUTHORS' CASES

Type of Trauma	No.
Auto accident	16
Fall	5
Football injury	2
Penetrating injury	1

Signs and Symptoms of Delayed Splenic Rupture. It is evident that the signs and symptoms of delayed splenic rupture will differ from those of acute rupture in that they develop relatively more slowly and insidiously, depending upon the rate of blood loss. The rate of blood loss may be very gradual indeed until such time as the "latent period of Baudet" (the period in which the body is able to tamponade the bleeding area adequately) is terminated by sudden severe intraperitoneal hemorrhage. In our series the duration of occult splenic injury and hemorrhage prior to diagnosis ranged from two to fourteen days in six of eight cases of delayed rupture; in the remaining two cases an even longer time elapsed prior to diagnosis or secondary hemorrhage. Hence there is usually adequate time for correct diagnosis prior to such hemorrhage, providing the following indications of splenic trauma are noted and their significance appreciated.

Evidence of Loss of Circulating Blood Volume. Although 75 per cent of our patients with delayed splenic rupture did exhibit borderline anemia on admission, in almost all cases such an anemia could have been explained on the basis of associated injuries. Because of these associated injuries, the leukocyte count was rarely of use to us in diagnosis. Bailey and Schreiber,[7] as well as Byrne,[3] have also tended to discount the value of this procedure. Because of the limited value of admission erythrocyte and leukocyte counts in delayed splenic rupture, a clear indication exists for establishment of a baseline of the patient's status by means of erythrocyte counts, and hemoglobin, hematocrit and blood volume determinations, so that repetition of such tests at intervals will clearly indicate whether or not gradual blood loss has occurred. This is the course we have followed and which we have found to be the most reliable indication for abdominal exploration.

Wright and Prigot[2] have recommended paracentesis as a more rapid method of detecting intra-abdominal hemorrhage; however, Dobson[5] believes this test is unreliable. We have had no personal experience with its use but do believe that misinterpretation of results would be common.

Evidence of Mass. On physical examination a mass is not always found in the left upper quadrant in delayed splenic rupture, but we have detected such a mass in three of our eight cases. Fortunately, splenic bleeding will more often produce one or more suggestive x-ray findings, such as: an ill defined, enlarged splenic shadow (Fig. 1); splinting of the left diaphragm; displacement of the stomach to the right producing a serrated greater curvature; downward displacement of the transverse colon; or, rarely, evidence of fluid between bowel loops.[4,5,8] However, many of these findings are difficult to recognize or evaluate, especially in the presence of severe contusion or fracture of the left chest. In three cases of delayed splenic rupture, in which barium studies were taken after injury and just prior to surgery, we were unable to demonstrate positively any of the aforementioned findings, even in retrospect. Byrne[3] has had similar difficulties in radiologic interpretation, although at times these roentgen findings may make the diagnosis of splenic rupture quite obvious.

Evidence of Peritoneal Irritation. Blood loss into the peritoneal cavity at first may be fairly well localized to an area beneath the ribs; yet even when it is not, its presence does not invariably produce sufficient parietal peritoneal irritation to cause significant pain. More

important, such pain if present may be overshadowed by pain due to local contusions or hemilateral lower rib fractures. Bowel sounds commonly remain normal in the presence of intraperitoneal hemorrhage, whereas muscle spasm or rebound tenderness is often absent or masked by associated injuries.

Of more diagnostic value are the signs produced by irritation of the diaphragmatic peritoneum. Such irritation will often produce pain referred to the left shoulder; or such pain may be elicited, according to Sagesser,[9] by pressure applied between the sternocleidomastoid and scalenus muscle of the left side. This diaphragmatic irritation, as previously noted, may cause limited excursion of the diaphragm, but again this finding is of limited value in the presence of lower rib fracture.

It is worth noting that Hinton and Steiner[10] encountered only two cases of splenic rupture (an incidence of 0.71 per cent) in a study of 279 patients with rib fractures. However, at the St. Francis Hospital from 1947 to 1953 we found 153 consecutive cases of fracture involving one or more of the lower left eight ribs; of these, six had splenic rupture, an incidence of 3.9 per cent. Perhaps the true incidence of splenic rupture is even higher than this but is not detected because of the efficiency of the body's ability to tamponade and to repair. In two additional cases which did not come to surgery localized pain, anemia and transitory shock compatible with a diagnosis of delayed splenic rupture were present. X-ray evidence of basal pleuritis on the left side in these two cases also strengthened the diagnosis, as discussion of this finding will illustrate.

Basal Pleuritis. A short time ago we observed a patient with delayed splenic rupture whose chest x-ray three days following injury and just prior to splenic rupture demonstrated marked left basal pleuritis and an associated minimal fracture of the left ninth rib. This basal pleuritic reaction was characterized by moderately increased diffuse density of the left lower lung field, most marked at the diaphragm and gradually disappearing one or two rib spaces cephalad. We theorized that this clouding might be the result of the influx of fluid or cells into the diaphragmatic pleura and nearby lung tissue as a local response to the accumulation of blood beneath the diaphragm from a damaged spleen. We therefore reviewed our other cases of delayed splenic rupture to determine

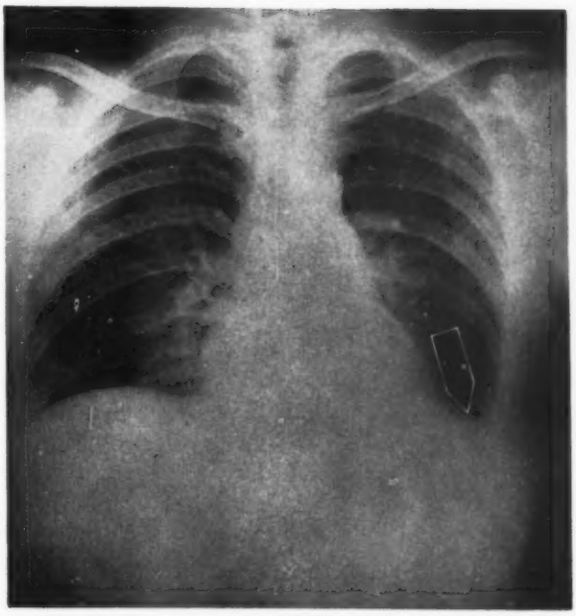

FIG. 1. Chest x-ray; basal pleuritis without associated rib fracture; splenic rupture occurring two days later.

whether or not such a pleuritic response was a common occurrence. We were surprised to note that whether or not rib fractures were associated this response was shown by all of our four patients with delayed splenic rupture who had had chest films taken more than twenty-four hours after injury. (Figs. 2 and 3.) Of the four additional cases of delayed splenic rupture subsequently reviewed at the two military institutions, chest radiographs were taken more than twenty-four hours after injury in only two; of these, one showed basal pleuritic response. Thus in five of six cases of delayed splenic rupture in which x-rays were taken twenty-four hours after injury a basal pleuritis was shown, an incidence of 83.4 per cent. The incidence of this pleuritic response in fractures of one or more of the five lower left ribs is of interest. Of 126 consecutive cases in which chest films were taken twenty-four or more hours after injury, such a response was demonstrated in nine. However, of these, four patients were found at surgery to have delayed splenic rupture and two, as already mentioned, had findings compatible with splenic injury; in the remaining cases, although splenic rupture may have been present, we were unable to make such a clinical diagnosis. Because of the relative frequency of this basal pleuritic response in cases of delayed splenic rupture, we believe it is a diagnostic sign of some importance

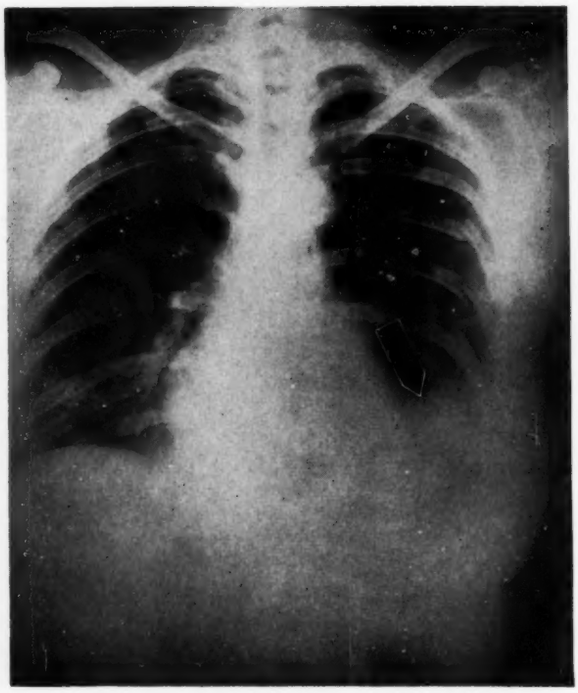

FIG. 2. Chest x-ray; basal pleuritic reaction with associated rib fractures; delayed splenic rupture occurring four days later.

FIG. 3. Splenic mass is seen in the left upper quadrant ninety-six hours after injury; delayed rupture occurring on the fifth day.

TABLE III
MORTALITY RATE OF SPLENIC RUPTURE

Author	No. of Cases	Over-all Mortality Rate (%)	Surgical Mortality (%)
Bessel-Hagen (1900)[11]	37	46	46
Buxton (1922)[12]	...	....	29*
Wright and Prigot (1939)[2]	30	43	27
Rousselot and Illyne (1941)[6]	17	29	29
Roettig et al. (1943)[1]	22	32	7
Byrne (1950)[3]	101	....	17
Larghero and Guiria (1951)[13]	18	6	6
Fowler and Bollinger (1954)	24	16.6	4.8†

* Reviewed surgical mortality of splenic rupture up to 1919; number of cases not stated.

† Three patients with acute splenic rupture died with skull fracture and multiple injuries prior to surgery; 1 patient with acute rupture died after operation due to failure of blood-clotting mechanism.

TABLE IV
MORTALITY RATE OF DELAYED SPLENIC RUPTURE

Author	No. of Cases	Total Mortality (%)	Surgical Mortality (%)
McIndoe (1932)[14]	46	39	27
Zabinski and Harkins (1943)[4]	70	16	16
Fowler and Bollinger (1953)	8	0	0

and deserves, if possible, corroboration and emphasis.

Mortality. Improvement of the over-all mortality of splenic rupture has been rapid in the past few years because of the greater availability of blood. (Table III.) However, this advantage is somewhat nullified by the disadvantage of multiple severe injuries due to high speed travel. Of our sixteen cases of acute splenic rupture, three patients died in the emergency room prior to surgery; these deaths were due to associated injuries, all of the patients having severe skull fractures and multiple injuries. Of the remaining thirteen cases of acute splenic rupture which came to surgery, there was one mortality not attributable to surgery. This patient, with suspected though undiagnosed aleukemic leukemia, had ruptured his spleen in a fall from bed. The patient survived surgery only to die postoperatively of

generalized failure of the blood-clotting mechanism. The mortality rate in delayed splenic rupture should be low since blood is available and only diagnostic accuracy is required. There were no deaths in the eight cases of delayed splenic rupture. The improvement in the over-all mortality rate of splenic rupture and delayed splenic rupture can be noted in Tables III and IV.

SUMMARY

1. The incidence of both acute and delayed splenic rupture is increasing due to high speed travel and now represents 0.15 per cent of all hospital admissions due to trauma.

2. The incidence of delayed splenic rupture is high, being present in eight of our twenty-four cases, an incidence of 33.3 per cent.

3. Splenic rupture is frequently associated with rib fractures. It occurred in 3.9 per cent of a consecutive series of 153 fractures of one or more of the lower left eight ribs.

4. Six of our twenty-four cases with splenic rupture had associated left lower rib fractures.

5. The establishment of a clinical baseline with respect to blood count, hematocrit and blood volume is clearly indicated. Frequent repetition of these tests is fundamental to the diagnosis of delayed splenic rupture.

6. The presence of a left basal pleuritic response as demonstrated by x-rays taken more than twenty-four hours after injury is highly suggestive of delayed splenic rupture. This response was present in 83.4 per cent of our cases.

7. Of sixteen cases of acute splenic rupture, three patients died soon after admission to the hospital because of multiple injuries including skull fracture. Of the remaining thirteen patients who came to surgery, all survived except one with aleukemic leukemia who died from his disease.

8. There was no mortality in eight cases of delayed splenic rupture.

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DISCUSSION OF PAPERS BY DRS. GLENN, FURSTE, KINNAIRD, GATEWOOD AND RUSSUM, AND BOLLINGER AND FOWLER

William L. Estes, Jr. (Bethlehem, Pa.): I will confine my remarks largely to the papers dealing with trauma to the liver, spleen and pancreas.

Our experience certainly parallels that of Dr. Glenn in the fact that injuries to the liver by blunt trauma certainly represent the area of greatest mortality in any series of non-penetrating abdominal injuries. I think it is due to the fact that in most cases there is very extensive hemorrhage. In trauma to the liver with severe hemorrhage it is frequently necessary to operate in the presence of shock, but invariably after proper measures have been inaugurated to treat shock, restore blood volume and improve the patient's condition. There should likewise be a very high incidence of suspicion of trauma to the liver in any questionable injury in the upper right abdomen when there has also been fracture of one of the lower ribs.

The only actual instance of injury to the bile ducts in our series was a complete avulsion of the gallbladder with laceration of the cystic duct. The clinical picture was very close to that of rupture of an upper abdominal viscus, a syndrome which demanded immediate operation.

In our series of twenty-five cases involving rupture of the spleen there were twelve cases of splenic rupture as an isolated injury, and all twelve pa-

tients survived splenectomy. However, in those cases in which there were concomitant injuries there were six deaths, three definitely due to the extra-abdominal lesion. There were six cases of delayed hemorrhage; two did not come to operation because the condition was not recognized. All were associated with multiple injuries elsewhere, and one particularly with severe cerebral trauma. When the patient is unconscious, the diagnosis of laceration of an abdominal viscus may be exceedingly difficult. Repeated examination at frequent intervals will be imperative. Again a high degree of suspicion of splenic rupture should be aroused if there is any injury to the ribs on the left side.

Lacerations of, or trauma to, the pancreas is of particular interest. We have encountered four such cases. In the first case a large hematoma which required drainage was found posterior to the head of the pancreas. Obstruction developed at the duodenojejunal junction but the duodenum was not ruptured. The obstruction was relieved by gastroenterostomy. Four weeks later acute suppuration of the gallbladder required cholecystectomy. Repeated attacks of vomiting and pain finally occurred, so that four years later the gastroenterostomy was replaced with a duodenojejunostomy. This procedure was necessary in order to correct the persistent duodenal obstruction. The patient has remained well (four years).

The second patient was a nineteen year old boy in whom, after two and a half months of conservative treatment for upper abdominal trauma, an upper right quadrant mass developed which proved to be a pseudo-cyst of the pancreas. The patient has remained well fourteen years following marsupialization of the cyst.

The third case was that of a woman who fell in a bathtub and injured her left side. She noticed an abdominal tumor within a few months but did not agree to surgery for ten years. The tumor proved to be a large pancreatic cyst. Marsupialization gave her relief for only six years. Recurrence of the cyst required cystenterostomy for cure. Two years later she died of carcinoma of the pancreas, originating in the cyst wall.

Internal drainage of these pancreatic cysts would seem preferable to marsupialization; but if pseudocyst of the pancreas develops in the tail area or left half, resection should be considered in the good risk patient because of the possibility of carcinoma occurring later in the remaining cystic remnant.

The fourth case was that of a man who died of diffuse peritonitis forty-eight hours after the suture of a large rupture of the stomach in the pars media. At autopsy, in addition, a retroperitoneal rupture of the duodenum was found as well as a large retroperitoneal hematoma which was caused by complete laceration of the middle third of the pancreas. This latter finding raises the question of whether hematoma in the pancreatic area found on abdominal exploration for trauma should be explored in order to avoid extensive damage to the pancreas going unrecognized.

Again I want to express my appreciation of this splendid symposium on the surgery of abdominal trauma.

GEORGE B. SANDERS (Louisville, Ky.): Although I wish to discuss the fine paper of Dr. Kinnaird, I hope my remarks will be found pertinent to the other splendid papers.

I would like to corroborate the statement about the value of epidural block in some of these cases. In our practice we have used continuous epidural block not only for the relief of the pain of pancreatitis, but also for the relief of pain in other acute conditions of the upper abdomen and for the relief of pain postoperatively in patients who have had extensive upper abdominal incisions or patients who are large, obese, bulky and perhaps hypersensitive to pain.

It is important to realize that the level of block age must be high enough to achieve the desired result in operations in the upper abdominal cavity; and in pancreatitis especially, the level should reach the fifth or sixth thoracic segment of the cord. With the solutions used by our anesthetists, that means the tip of the catheter has to lie at about the seventh or eighth dorsal vertebra.

I would also like to say that I am not at all sure it is wise to so departmentalize the various injuries following acute abdominal trauma as we have done at this meeting.

The early diagnosis of these conditions so often rests upon what one finds when the abdomen is actually opened. It is necessary, therefore, to make the decision and operate upon these patients very often on the merest suspicion that associated abdominal injury exists or could exist, without the benefit of certain more or less specific diagnostic tests.

I would like to refer to a paper given by Mr. Rusco Clark of the Birmingham Accident Hospital, Birmingham, England, in which he presented 107 consecutive abdominal laparotomies for closed abdominal trauma; these patients were operated upon on the merest suspicion that an abdominal injury of serious degree might co-exist.

Of those 107 cases, eighty-three patients survived. In these eighty-three there were twelve negative laparotomies; also there were twelve positive laparotomies, in which case nothing more could be achieved by surgery once the abdomen was opened.

In the twenty-four patients who died, there was one negative laparotomy; however, it is not conceded that this patient died as the result of the laparotomy or from its effects. The other twenty-three patients died either of severe intra-abdominal

injuries for which little or nothing could be done or of very severe extra-abdominal injuries for which nothing could be done.

Laparotomy was not implicated in the cause of death in any instance.

I will quote directly from Mr. Clark's paper because it epitomizes certain points which I think are of particular value to this Association.

Mr. Clark says: "In practice it is essential to bear in mind simultaneously a large variety of possible traumatic lesions and pathological sequelae. Hesitation to advise surgery or to seek the advice of experienced consultants may arise from an excessive desire to make a detailed diagnosis during the preoperative phase. This can be understood in the light of an approach to the teaching of surgery which treats lesions of intra-abdominal viscera as separate entities with individual clinical features capable of being dissociated one from another. In practice we are presented not with 'a case of ruptured spleen' but with a patient who may have an abdominal injury which may be dangerous if left alone.

"My experience in the care of such injuries suggests that their diagnosis and treatment cannot be separated from the diagnosis and treatment of other injuries. Traumatic surgery is not an anatomical specialty and owes its existence as a form of division of labor to a need for a specialized tempo. Neither is it just a matter of acute orthopaedic or plastic work. Its exponents must remain in contact with developments in a variety of fields of medicine and surgery, particularly those concerned with non-traumatic emergencies."

Everett P. Coleman (Canton, Ill.): I would like to make two brief comments in reference to two of these papers.

In regard to Dr. Furste's paper, some years ago I reported four cases of hematoma of the abdominal wall under the title of "Spontaneous Hemorrhage into the Rectus Sheath." All four of these cases came to surgery and they all had certain common symptoms.

First of all was the symptom of external abdominal pain. All patients were middle-aged individuals with a severe or persistent cough. Apparently the cough was a big factor. All had moderately severe pain; one patient had rather severe pain. We believed that the pain was caused by the following fact: Since the artery ran posterior to the muscle, the blood was in contact with the anterior surface of the peritoneum; and since blood inside the peritoneum causes pain, we believed that this irritation on the outside probably produced the same symptom.

All of these patients were anesthetized by the same nurse anesthetist. About two years ago one of my associates was called at about 4:00 A.M. by this nurse, who told him, "I am having some pain. I think I need a hypo, and I think should be taken to the hospital because I am sure I have spontaneous hemorrhage into the rectus sheath." (This is a new procedure, having a patient make the correct diagnosis for the physician.) The patient had had a cough during the night and the hemorrhage had developed. It was rather extensive and the pain was moderately severe.

One comment in reference to the paper by Drs. Bollinger and Fowler: The authors spoke about delayed splenic rupture going from two to fourteen days after injury. I would like to mention briefly one case which went twenty-five days following injury. A young man was in an automobile accident and sustained the usual steering wheel injury and also some trauma to the chest. According to x-ray and clinical findings, there were no broken ribs. A rather severe laceration of the neck was repaired by the patient's doctor, yet the patient continued to have rather progressive anemia with no apparent or obvious abdominal symptoms.

I saw the patient in consultation five days later. His neck wound was healing; he had just had another transfusion and seemed to be in pretty good condition. The abdomen was rather doughy but there were no particular findings. Nothing specific could be determined.

The patient went home on the fourteenth day, and was up and about, apparently in good condition.

On the twenty-fifth day he had severe abdominal pain and went into shock. I saw him after he had been hospitalized for five or six hours. He was in poor condition and was being given blood under pressure. We questioned whether the condition was a splenic rupture or leakage from a previous rupture of the liver. A transverse incision was made. I have never seen so much blood in the abdomen in one person. Apparently all of the blood the patient had received had gone out slowly through the splenic laceration. There was a sudden release of pressure for some reason, and rather severe hemorrhage. Splenectomy was performed and recovery followed.

J. Dewey Bisgard (Omaha, Neb.): Dr. Gatewood asked me to discuss his paper so I will limit my remarks to that article. I want to commend Drs. Gatewood and Russum for their excellent discussion of a very important, though unusual, subject. It is important to know that the appendix is vulnerable to non-penetrating forces of injury and that the appendix should be inspected in the course of exploring a traumatic abdomen. I think, however, there are also other important implications in this paper.

First, I want to call attention to the medical-legal question which is often involved, and second, I want to speak of masking in trauma, illustrated so well in the authors' second case.

Since injury so often involves legal responsibility and compensation, the question of the relationship of a given lesion to trauma becomes important. In the first case this relationship seems definite—an obvious example of multiple injuries with traumatic amputation of the appendix. In the second case, however, the relationship, it seems to me, is open to question. Did the perforation of the appendix which was followed by peritonitis and death result from the force that fractured the hip, or was it the result of a coincidental attack of acute appendicitis? I merely wish to point out that this question is of more than academic interest and importance in cases which become involved in litigation.

There are references in the literature which indicate that the appendix can be traumatized in injury.

The second implication refers to the errors in diagnosis or, may I say, omissions in the complete evaluation which not infrequently occurs in cases of multiple injuries. I am sure that everyone who takes care of many of these cases has overlooked, for a short time at least, one of multiple injuries because the picture was confused or obscured by such dominating symptoms as stupor from head injury and shock. Also, it is very easy to misinterpret the source of symptoms, as in the authors' second case. Abdominal distention, pain and tenderness occur frequently as a result of the retroperitoneal hemorrhage associated with injury to the spine and pelvis, and may raise some doubts regarding the presence of an intra-abdominal lesion or serve to mask such a lesion.

Recently I saw a patient, stuporous from a head injury, who was suffering severe dyspnea from an associated tension pneumothorax. For a time the dyspnea had been attributed to the head injury and the pneumothorax overlooked altogether.

Some errors in these cases are probably inevitable, but they can be minimized by complete examinations, constant observation, frequent reexamination and earlier exploratory investigation. Unless a patient is critically ill, simple exploration involves little or no risk.

One cannot repeat too often the dictum, "Explore upon suspicion." If one procrastinates until the issue is obvious, too often he waits too long.

J. Huber Wagner (Pittsburgh, Pa.): Dr. Bisgard has discussed some of the things I intended to mention in regard to Drs. Gatewood and Russum's paper, originally presented as "Traumatic Appendectomy and Traumatic Appendicitis." I think the terms here are wrong. The authors, in the first case, showed an injury to the appendix. In fact, it was a rupture, a complete transection; hence I think the term appendectomy is wrong. The appendectomy was later performed by Dr. Gatewood, i.e., removal of the appendix.

The second case report, to me, did not in any way prove that the injury was a causative factor in this condition. I also think it was coincidental.

We have treated many fractured hips. There are no associated injuries to the appendix. In this case, from the symptoms, none of them showed any trauma to the abdomen or the abdominal wall.

In my opinion, the appendicitis developed coincidentally with the injury. To have this inference accepted by this society, I believe, would be wrong, because it infers that an injury causes what we accept ordinarily as an inflammatory condition of the appendix. Trauma to the appendix, yes, but to assume or accept an abdominal injury as the inciting factor of an infectious inflammation to a single organ, I think, is wrong.

I do not believe this Association should accept this paper with the title under which it was originally presented.

Fraser N. Gurd (Montreal, Canada): I would like to discuss Dr. Fowler's paper and to introduce a differential diagnosis to be considered in steering wheel injuries and other trauma to the thorax. This is the diagnosis of myocardial contusion. When rupture of the spleen is under consideration and when a competent cardiologist suggests that the entire clinical picture may be due to myocardial injury, the surgeon faces a dilemma.

To cite one illustration, a man in his sixties was admitted to the Montreal General Hospital after suffering a severe blow to the left anterolateral portion of the chest. He complained of severe pain in the left side of the chest, and substernal and upper abdominal pain. He had low blood pressure, hemoglobin of 60 per cent and some upper abdominal tenderness; the electrocardiographic changes showed fairly severe myocardial injury of an acute nature. After much discussion we agreed to keep him in bed for two weeks; the cardiologist was waiting for the heart to recover while I was hoping that the spleen "would behave itself."

On the fourteenth day the patient got out of bed and immediately collapsed. However, at this time the hemoglobin fell below 50 per cent; transfusion was therefore given and the patient was explored. A complete rupture of the spleen was found and splenectomy was performed, from which he recovered nicely.

For the benefit of residents, I might add that in delayed cases two or three weeks old the rigidity of the splenic pedicle due to organizing hematoma may render splenectomy somewhat more difficult than is ordinarily the case.

Myocardial contusion is an entity which is becoming increasingly recognized and which may indeed be severe enough to add to the hazard of incidental surgery. In such cases showing a strong suspicion of intra-abdominal injury it takes some strength of character to proceed to exploration in

the face of an expert opinion that the heart has been badly bruised.

ROBERT MAYO TENERY (Waxahachie, Texas): I would like to report one case which can be added to Dr. Glenn's paper.

Overseas we saw a young soldier with a perforating wound of the abdomen who on first exploration showed only perforation of the stomach. This was repaired. Subsequently a definite syndrome developed wherein the patient would suffer severe shock, followed by pain in the right upper quadrant and then jaundice and melena. There was no hematemesis. Each attack required 2,000 to 3,000 cc. of blood to bring him out of shock, and he had about seven such episodes over a period of two months. Finally he had one episode with very severe hemorrhage and he died.

The diagnosis was made before the patient's death and was confirmed at autopsy, which showed traumatic aneurysm of the hepatic artery extending into the right hepatic duct. Intermittently this aneurysm would bleed and a clot would form in the common duct, producing jaundice which would gradually clear when the clot passed out of the duct.

WALTER L. MERSHEIMER (New York, N. Y.): I would like to add a few remarks in discussion of Dr. Furste's paper. In recent years we have observed three patients who were operated upon and in whom hematoma of the rectus muscle was found.

We did not make a correct preoperative diagnosis in any instance, although it was suspected as a possibility in one case. Preoperative diagnoses made were: first, acute cholecystitis; second, torsion of an ovarian cyst; third, diverticulitis of the sigmoid colon with perforation and abscess formation. In no instance was a history of trauma available.

All of our patients were women. Two of them were elderly, and in these two women hypertensive cardiovascular disease was present. All three patients apparently had spontaneous rupture of epigastric vessels as found at operation. In the two patients with hypertensive cardiovascular disease the microscopic examination of the epigastric vessels did show advanced arteriosclerotic changes. We believe, therefore, that the etiologic factor may frequently be primary vascular disease without trauma or the trauma of secondary significance.

PRESTON A. WADE (New York, N. Y.): I think that after the presentation of all these very fine papers we should make some reference to the prevention of some accidents which are seen. Evidence has shown that the use of seat belts will on many occasions prevent abdominal injuries in automobile accidents. This has not been emphasized in any of the foregoing papers, and yet we know that there is evidence, as shown by John Moore's research, that many of these accidents may be prevented by the use of such belts.

I might point out that this society went on record to recommend that seat belts be advised and that the automobile manufacturers be asked to consider them in new cars. I would also like to point out that in the new Ford car you will find all of the safety devices which this organization and the College of Surgeons have advocated.

I would just like to say a word in reference to Drs. Gatewood and Russum's paper. Dr. Wagner may be quite right and I agree with him in some respects. The second case of appendicitis may very well have been a coincidental finding and not the result of the trauma. However, I do believe that the authors have a right to their opinion, and so I would like to say that this society should accept the paper and not try to judge or suppress it.

LOUIS C. ROETTIG (Columbus, Ohio): We have not reviewed our cases of delayed rupture of the spleen since the report which I gave before this organization in 1942. However, we have seen quite a number of these cases since that time. I would just like to re-emphasize a very valuable observation which Drs. Bollinger and Fowler have made, namely, the presence of signs of irritation above the left diaphragm.

In almost all of the cases of delayed splenic rupture which we have seen since 1946 we have found that immobilization of, or the evidence of fluid above, the diaphragm on the left side has been a very frequent and predominant finding.

I think it is highly timely that this will appear in the literature with the publication of Drs. Bollinger and Fowler's paper.

FRANK GLENN (closing): I would like to make a plea in the consideration of abdominal trauma: In the future, those of you who are interested and have the facilities available, search out in your pathology departments (either in your hospital or medical examiner's office) patients who have died who had exhibited massive trauma of the abdominal contents.

I am convinced that the greatest contribution is going to come from emphasizing exactly what has happened in a number of patients who could have been saved if they had undergone surgery. I know of no better way of finding this out than to bring our deficiencies before the profession. This will probably be more fruitful than reporting good results from our operative attempts.

WESLEY FURSTE (closing): I think Dr. Coleman should be congratulated for having such an observing anesthetist.

As I have already stated, in eighty-four reviewed cases, the correct diagnosis was made in only about one-fifth.

Our main purpose in presenting the paper was to emphasize the fact that such a condition can occur, so that we will consider it preoperatively.

DAVID W. KINNAIRD (closing): I would like to underscore what Dr. Glenn and Dr. Sanders and others have said. Although we were primarily interested in reporting injuries to the pancreas alone, if there is any suspicion of other serious, associated intra-abdominal injuries, exploration should be done immediately.

JOHN W. GATEWOOD (closing): In answer to Dr. Wagner, I think that our second case certainly is open to question. I think it is of value from the standpoint that I firmly believe appendicitis can be caused by trauma. In this particular case there is no absolute way to prove it. I think the cases are rare. Dr. King reports a case with division of the mesentery of the appendix in which suppuration developed.

Under those circumstances the condition could be proved absolutely. In this case Dr. Wagner is completely correct; the case is open to question.

EDSON F. FOWLER (closing): I wanted to comment briefly on Dr. Furste's paper. I have seen at least three cases of spontaneous hematoma in the abdominal wall which have occurred during pregnancy and which have been absolutely indistinguishable from acute appendicitis until the time when we made our incisions. I therefore do not think it is a very uncommon entity.

I would like to thank the discussers of our paper on delayed splenic rupture, particularly Dr. Roettig who has been interested in this problem for some time. He has seen what we have observed in x-ray films, namely, the basal pleuritic reaction. Since it has not been emphasized previously, we were not quite sure whether we were really seeing something or talking ourselves into it.

The Operative Treatment of Penetrating Wounds of the Chest

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ONE sultry morning in the spring of 1944 an infantryman jumped from a landing craft and waded rapidly ashore in one of the island invasions in the Pacific. His usefulness to the Armed Forces was quickly terminated. As soon as he reached the beach, the nearby explosion of a mortar shell sent a small fragment into his chest, penetrating through the left seventh interspace in the mid-axillary line.

He was evacuated to a hospital ship where an x-ray of his chest was taken, revealing a small amount of fluid and a minimal degree of pneumothorax. After his initial shock he was not in any distress. The following day thoracentesis was performed and 400 cc. of blood withdrawn. Thereafter, aside from chemotherapy he received no further treatment, and recovered rapidly.

Three weeks later he was put ashore for reassignment. While picking up his duffle bag and slinging it over his shoulder he felt a sudden pain in his left shoulder, became short of breath and fainted.

The number of psychiatric examinations and cursory physical examinations which he had thereafter will not be mentioned. Finally, a surgeon took an x-ray of his chest and quickly ordered an upper gastrointestinal series which revealed a ruptured left hemidiaphragm.

The story of this infantryman is not part of our present case material, but it started us toward a more positive and dynamic approach in the management of penetrating wounds of the chest. It illustrated the first cardinal point of our present concept.

Although an accurate history is obtained and physical and x-ray examinations are carefully done, these can never be an infallible guide to the extent of trauma inside the thorax. An exception to this statement may be in cases of stabbing when the weapon is brought to the doctor. Even then, only the maximum depth may be known; the direction and extent of laceration can only be surmised. In the case of either stab wounds or bullet wounds the damage at the point of entrance is no foundation at all upon which to form an opinion of the extent of the internal injury. One oscillation of a knife or an ice pick, or the ricocheting or the splattering of a bullet as it strikes bone, may increase the internal injury many times over the small laceration seen in the skin. Large vessels may be involved, and death may occur rapidly.

A brief review of cross sectional thoracic anatomy will quickly help one realize the importance of direction and depth. When penetrating chest injuries result in hemothorax, pneumothorax or hemopneumothorax, operation should be performed. It is important to remember that when only pneumothorax is evident on the first x-ray, up to 400 cc. of blood may be present although not apparent. Penetrating injuries by a penknife or ice pick without hemothorax or pneumothorax may be treated expectantly.

The next point we wish to make concerns the obvious disregard held for thoracic surgeons by those people who inflict penetrating chest wounds; they lack the courtesy to plan their crimes at the front door of a large medical center. Penetrating chest wounds are such emergencies that they demand the immediate and skillful attention of the first available qualified surgeon. We therefore believe that any qualified surgeon should be able to manage this problem and there should be no delay for the purpose of transporting these patients to a thoracic surgeon.

In treating a penetrating wound of the chest the surgeon is immediately confronted with the decision of whether to operate or treat expectantly. Expectant management with its many uncontrollable hazards, its imponder-

TABLE 1

PENETRATING WOUNDS OF THE CHEST WITH HEMOTHORAX AND/OR PNEUMOTHORAX

Case No.	Type of Penetration	Type of Trauma	Morbidity Expressed in No. of Days in Hospital		Remarks and Complications
			Operated	Not Operated	
1	Bullet	Hemothorax	..	41	Persistent temperature elevation
2	Bullet	Hemothorax	..	Died	In hospital 4 hr.; could have been operated upon and saved
3	Knife	Pneumothorax	6	.....	Laceration of lung
4	Knife	Hemothorax	10	.....	Laceration of lung
5	Knife	Hemothorax	11	.....	Bleeding, intercostal artery
6	Knife	Pneumothorax	..	5	.....
7	Knife	Hemothorax	10	.....	Laceration of lung and internal mammary artery
8	Bullet	Pneumothorax	..	8	Released with 35 per cent pneumothorax
9	Bullet	Hemopneumothorax	..	Died	In hospital 5½ hr.; buckshot in pericardium; lung not penetrated more than 3 cm.
10	Ice pick	Pneumothorax	..	6	.....
11	Bullet	Pneumothorax	7	.....	Laceration of lung
12	Bullet	None	..	6	.....
13	Knife	Hemopneumothorax	..	3 plus?	Hospitalized elsewhere; 3 wk. as outpatient
14	Ice pick	Pneumothorax	..	4	3 wk. convalescence
15	Bullet	Hemothorax	..	7	Fibrothorax developed
16	Knife	Hemothorax	..	10	.....
17	Bullet	Hemopneumothorax	..	17	.....
18	Bullet	Hemopneumothorax	..	75	Thoracotomy on 11th day; empyema developed; drained on 57th day
19	Knife	Pneumothorax	..	9	.....
20	Knife	Hemothorax	..	11	.....
21	Bullet	Hemothorax	..	7	.....
22	Bullet	Hemopneumothorax	..	25	Persistent hemopneumothorax
23	Bullet	Hemothorax	..	49	Empyema developed
24	Knife	Hemopneumothorax	6	.....	Laceration of lung
25	Knife	Pneumothorax	..	8	Released with pneumothorax
26	Bullet	Hemopneu mothorax	..	8	Released with pneumothorax

TABLE 1 (*Continued*)

Case No.	Type of Penetration	Type of Trauma	Morbidity Expressed in No. of Days in Hospital		Remarks and Complications
			Operated	Not Operated	
27	Bullet	Hemothorax	..	9	.....
28	Knife	Pneumothorax	..	5	Released with pneumothorax
29	Knife	Pneumothorax	..	2	.....
30	Knife	Hemopneumothorax	11	.....	Laceration of lung and intercostal artery
31	Bullet	Hemopneumothorax	10	.....	Laceration of lung
32	Knife	Hemothorax	5	.....	Laceration of lung
33	Knife	Hemothorax, cardiac tamponade	17	.....	.....
34	Knife	Hemopneumothorax, hemopericardium	14	.....	.....
35	Knife	Hemothorax	8	.....	.....
36	Knife	Hemothorax	8	.....	.....

ables of diagnosis and its later complications may or may not restore the patient to a normal physiologic status. Operation eliminates the imponderables of diagnosis and many of the complications, which we will discuss later, and decreases the average morbidity to a generally predictable short interval of time.

The diagnosis of hemothorax and pneumothorax can usually be made by percussion and auscultation. An x-ray taken later may often be needed only for confirmation. The emergency care of these patients is very important and must be undertaken without delay. Large gaping wounds in the chest wall should be closed, taking care not to set up a tension pneumothorax. The palm of the hand at first, a thick pad of gauze or a sheet or rubber cut from a rubber glove taped in place over the opening will serve as a temporary measure and prevent further collapse of the lung and the entrance of contaminants into the thorax.

Tension pneumothorax may be present and should be relieved as soon as possible. This may be done by inserting a large gauge needle into the chest, placing one end of a rubber tube over the hub of the needle and dropping the other end under water below the patient. Sterile tubing, although desirable, is not necessary in emergency care. The pressure of tension pneumothorax should always be released slowly to prevent the mediastinum from shifting too suddenly.

Shock must always be looked for and treated promptly. Whole blood should be given as soon as available. A plasma expander may be used until the transfusion is obtained.

X-rays should be taken when the patient is in condition to tolerate being moved. Underwater drains have been placed in patients suspected of having pneumothorax when no pneumothorax was present. X-rays will prevent such mistakes.

Conservative treatment consists of observing the vital signs, performing repeated thoracenteses for removal of air and blood, and administering antibiotics. A better method is the establishment of a closed thoracotomy attached to an underwater drain. If the hemothorax or pneumothorax is small at the time of the first x-ray it may be left entirely alone. Repeated follow-up x-rays must then be taken. This approach is fraught with complications. In

instances of pneumothorax, atelectasis is likely to occur if the re-expansion of the lung is delayed too long. Atelectasis may often be followed by pneumonitis, with the possibility of subsequent empyema. If the pneumothorax is permitted to remain too long, complete re-expansion of the lung may fail to occur. In the conservative treatment of hemothorax the possibility of massive hemorrhage and subsequent fibrothorax must also be considered.

Operative treatment consists of opening the thorax and correcting the injury. This is ideally done under general intratracheal anesthesia, but any competent anesthetist can manage, if need be, with a closed system. Because of the rapidity at which blood may be lost, whole blood should be immediately available if needed.

The usual operative approach is through a posterolateral incision, entering the thoracic cage through the fifth or sixth interspace. The ribs are spread without transection, thus decreasing the postoperative morbidity. When the presence of a heart wound is suspected, an anterolateral approach through the fourth interspace is used, and thorough exploration of the chest is performed. Lacerations of the great vessels are sutured when possible, bleeding vessels are ligated, and lacerations of the lung or other organs are sutured. Rarely lobectomy, more often segmental or wedge resection, may be necessary. Before closure, the wound of entrance is débrided and the chest is thoroughly irrigated with warm normal saline solution. A Pezzar catheter is placed for drainage for forty-eight hours.

To evaluate the results of the operative as opposed to the non-operative management of penetrating wounds of the chest, we first reviewed over 100 cases from the services at White Cross Hospital and Ohio State University Hospital in Columbus, Ohio. The ten-year period from 1944 to 1954 was selected because during that time the present day technics of anesthesia and antibiotic therapy were all available. However, after our first review it became apparent that a further selection of cases would have to be made. Only uncomplicated penetrating wounds of the chest were selected. Cases showing other associated major trauma were discarded since these factors would influence morbidity and mortality rates. Finally, thirty-six comparable cases were selected. This degree of comparable trauma was the only criterion for selection. Thirteen cases were treated by operative management. The remaining twenty-three cases were treated conservatively with the insertion of a catheter and underwater drainage. It can be noted in Table 1 that in the non-operative group there were two deaths from laceration of large pulmonary vessels. Both patients were in the hospital long enough (four and five hours, respectively) so that if operation had been elected, the lacerations could have been repaired and the patient saved. The following two comparable cases were included in the operative group.

Case 2. A thirty-two year old man was admitted to the hospital after being stabbed in the back. He had, in turn, shot his assailant in the abdomen. Both arrived together. The patient with the abdominal wound, considered to be more critically injured, was taken to the operating room immediately. The stab victim was ambulatory and was sent to the Radiology Department for chest x-rays which revealed hemopneumothorax. He was placed in bed. Thereafter he was not observed as frequently or carefully by the house staff, who were preoccupied with operation on the other victim. The stab victim soon went into shock. This could not be corrected and he died in four hours. Autopsy revealed a laceration of the inferior pulmonary vein.

Case 31. J. S., a twenty-five year old white woman, was shot in the back with a .38 calibre revolver by her husband twenty minutes before her arrival at the Emergency Room. The bullet had entered the fourth interspace posteriorly and passed through the chest to lodge subcutaneously in the left breast. While being examined by the resident, her breath sounds disappeared and the entire chest became dull to percussion. Her blood pressure dropped precipitously. Plasma was started immediately. She was taken directly to the operating room where transfusions were started. The chest was opened, revealing a laceration of the base of the left upper lobe and of the branch of the pulmonary artery to the lower lobe. The wall of the vessel was sutured. The lung was débrided and sutured to prevent air leak. The patient made a completely uneventful recovery and was discharged on her tenth postoperative day with complete re-expansion of her lung.

Of the twelve cases treated by immediate operation, the maximum hospital stay was seventeen days; the shortest was five days. The two patients with the longest hospitalization, seventeen and fourteen days, respectively, both had cardiac as well as pulmonary lacerations. There was no mortality in this group. The

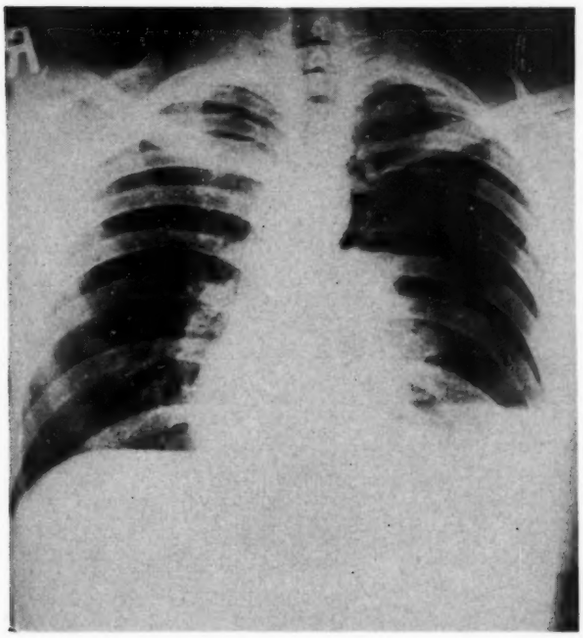

FIG. 1. Case 24. Chest x-ray on admission. Patient had stab wound of the chest with hemopneumothorax and laceration of the lung; treated operatively.

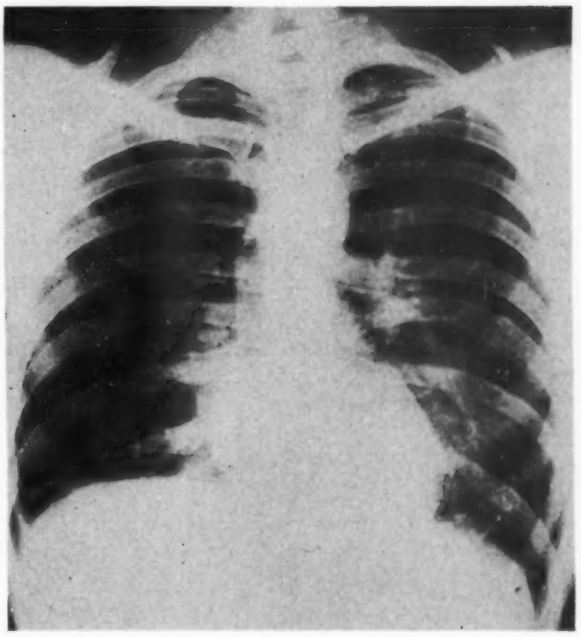

FIG. 2. Same case as Figure 1. Thirty days after patient's discharge from hospital. X-ray shows no evidence of fibrothorax and complete re-expansion of lung.

average morbidity in terms of hospital stay was nine and a half days. On leaving the hospital these patients showed no limitation of motion and lung re-expansion was complete. Fibrothorax did not develop in any of these patients. (Figs. 1 and 2.)

Twenty-three cases were treated conservatively. Two of these patients died five and four hours, respectively, after admission to the hospital. Neither patient was taken to the operating room. Both had lacerations of large pulmonary vessels and could probably have been saved by immediate operation. They represent errors in judgment on the part of both the attending and house staff. Time was wasted in an attempt to get these patients out of shock. These measures should have been performed on the operating table. These cases were included as a comparison to two in the operated group who survived. They also illustrate the fact that many of these chest wounds are cases of extreme emergency. If the patients are to be saved the first surgeon to see them must consider intervention and quickly make the decision for operation.

Empyema developed in two of the cases treated conservatively. Because of this their hospital stay amounted to seventy-five and forty-nine days, respectively. Eventually both had to be operated upon for the empyema.

CASE 18. A nine year old white boy was accidentally shot in the left chest. The bullet entered the chest in the fifth interspace, 2 cm. lateral to where cardiac dullness ended. The entrance point was ragged and powder-stained. The point of exit was the tenth left posterior interspace just lateral to the spinous process. Admission x-ray showed hemothorax. Thoracentesis was performed two days after admission. On the eleventh hospital day, because of persistent hemothorax, thoracotomy was performed and a bleeding intercostal vessel was ligated. The patient persistently ran a spiking temperature and on the sixty-second hospital day a portion of a rib was resected and an empyema cavity was drained. The patient was released from the hospital seventy-five days after admission.

This complication of conservative management can, does and will continue to occur, in spite of antibiotics, when the intrathoracic wound is not débrided and cleansed. Compared to the maximum hospital course of seventeen days for the operative group, this patient's stay represents a tremendous loss of time and money to the patient. A return to a completely normal-functioning lung is almost impossible.

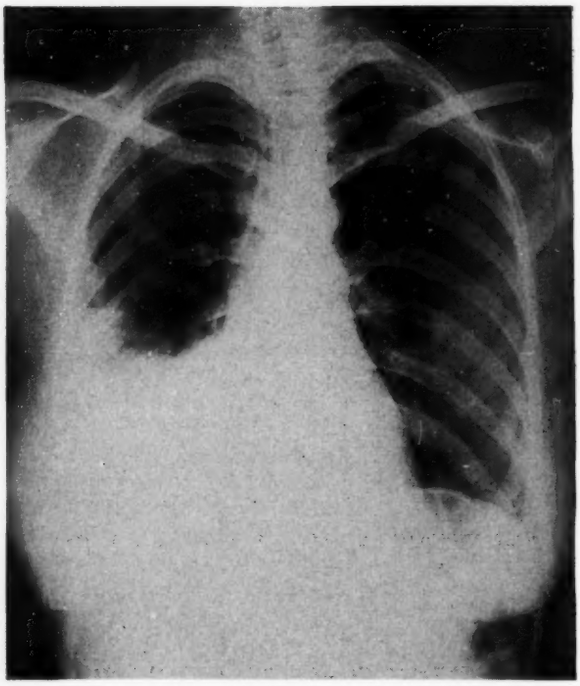

FIG. 3. Case 15. Patient admitted with a bullet wound of the chest and hemothorax; treated conservatively. Patient was discharged in seven days with fluid still present in the chest.

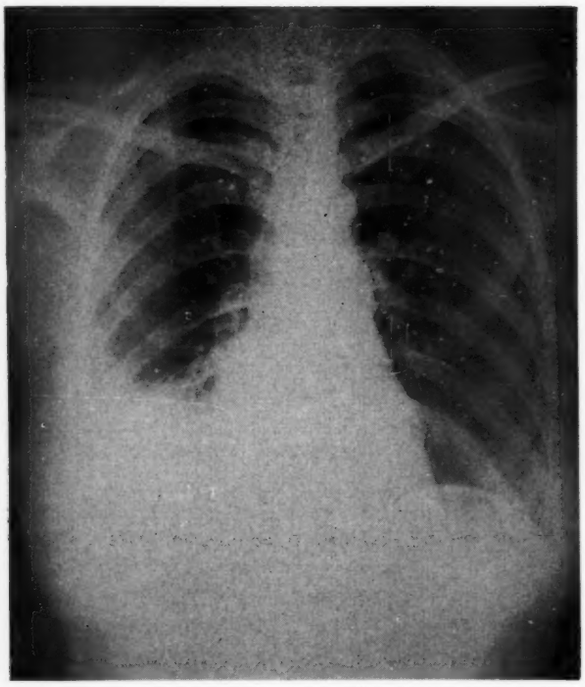

FIG. 4. Same case as Figure 3. Chest x-ray of patient thirty days postoperatively showing early fibrothorax developing on the injured side.

Four patients in the group receiving conservative management were released with a partial hemopneumothorax still present. Three of these patients were hospitalized for eight days; the fourth, for five days. They should have remained until lung re-expansion was complete. This would have further increased the morbidity of the non-operative group. This type of discharge is no longer acceptable. (Figs. 3 and 4.)

The average morbidity for the non-operative group, as expressed in hospital stay, was fifteen days.

Twelve cases were treated by immediate operation. The maximum stay for this group was seventeen days. The next longest was fourteen days. These two patients had both heart and lung lacerations. The shortest stay was five days for a patient with laceration of both lung and right diaphragm. The average morbidity for this group, in terms of hospital stay, was nine and a half days. There was no mortality in this group despite the fact that two cases of laceration of major vessels were included here, too. One of these has been reported (Case 18).

The following case illustrates how easily a major laceration of the heart could be overlooked, had not the operative approach been used.

CASE 34. A twenty-three year old white man was admitted to the Emergency Room following a knifing. When first seen he had a clean, incised wound, gaping and sucking air, through the left fifth interspace anteriorly, extending from the edge of the sternum almost to the anterior axillary line. He was not, however, in shock and there appeared to be very little bleeding. The wound was so clean and the damage appeared so slight that the resident at first wanted to suture it in the Emergency Room. The patient was taken to the operating room where the wound was enlarged, revealing a long laceration of the pericardium and a smaller, but deep laceration of the left ventricle near the apex. Well over 500 cc. of blood lay concealed and clotted in the diaphragmatic gutter posteriorly. A laceration of the lingula was also present. The lung, pericardium and myocardium were sutured. The patient was discharged with complete re-expansion of the lung on the fourteenth postoperative day.

Empyema or fibrothorax did not develop in any patient in the operated group. All were dismissed from the hospital with an excellent result in terms of restored function.

Since the number of cases presented here is relatively small, we do not presume to draw any sweeping conclusions. However, the cases were carefully and fairly selected. We do not assert that every penetrating wound of the chest should be subjected to thoracotomy. The individual surgeon must make this decision. We do urge a more radical approach to this decision. Most of these patients should be explored by the first qualified surgeon who sees them. The intrathoracic damage will be found to be far higher than expected. This is in contradistinction to subcutaneous laceration of the lung from a fractured rib where the depth and direction of penetration is predictable. Operation avoids the imponderables of diagnosis. It reduces and should eliminate the complications encountered in conservative management. It thereby restores the patient to normal physiologic status. In addition to lower morbidity, operation has a lower mortality. We believe that these factors make operative management the treatment of choice for these wounds.

SUMMARY

Thirty-six cases of comparable penetrating wounds of the chest are presented. Thirteen were operated upon and twenty-three treated conservatively.

The operated group had no mortality and an average morbidity of nine and a half days, a maximum of seventeen days. There were no complications and all were returned to a normal physiologic status.

The unoperated group had two mortalities. The average morbidity was fifteen days with a maximum of seventy-five days. The complications of empyema and fibrothorax, which occurred frequently in this group, are discussed.

Penetrating wounds of the chest entail far greater intrathoracic damage than can be readily diagnosed by any available method short of open operation.

These wounds are of such severity that the first available qualified surgeon should be able to treat them.

An Analysis of the Late Results of Thoracoabdominal Wounding*

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THORACOABDOMINAL wounding has been a source of considerable concern to both the military and the civilian surgeon for many years. From the days of the Crimean War when adequate records were first kept it has been known as a serious wound where the fatality rate is high and the complications are severe.

The improvement of surgical approach and a better understanding of the immediate treatment of the wounded person has done much to reduce the early fatality. How this rate has fallen is well recorded, and it seems useless to burden the reader with such historic detail. However, strangely enough little has been written concerning the later complications of such wounding. It is thus the function of this report to record what happens to the patient in the later stages of recovery.

MATERIAL

This report deals with 142 patients who received wounds of the chest, diaphragm and abdomen as the result of one missile. Concomitant wounds of the chest and abdomen which injured the diaphragm are not included in this series. Of the patients reported, 112 were wounded in combat and 30 had wounds received in civilian life. Table I reveals the incidence of wounding on each side. In civilian life this incidence is much higher on the left than on the right, whereas in combat the relative incidence has no significant statistical value as the sides are almost equally divided.† The difference in complications of the two sides will be considered. Table II is a compilation of the incidence of visceral injury at the time of wounding. Table III lists the complications which occurred in the two series. In Table IV, which gives the results of treatment, the deaths are listed as one in thirty civilians, and two in a group of 112 in military service. These deaths occurred after the fifth day of wounding, which is the basis of time interval in this report. It will be noted that twenty-four civilians had primary recovery, which means that their recovery was uncomplicated and complete. Sixty-two in military service made primary recovery and were returned (as far as the chest and abdomen were concerned) to either active or limited duty within the theater of operations. Delayed recovery occurred in five civilians and in forty-eight of those in military service. These patients, as far as military service is concerned, were sent to the zone of the interior either because of other wounds or because it was thought that they would be of no further use in the theater of operations.

TABLE I

Side Wounded	Civilian Life	Military Service
Right	3	62
Left	27	50
Bilateral	0	8

TABLE II
INCIDENCE OF VISCERAL INJURY

Injury	Civilian Life (30)	Military Service (112)
Penetration of diaphragm only	6	14
Lung	3	45
Spleen	13	24
Stomach	11	15
Liver	4	56
Kidney	2	16
Small bowel	2	7
Colon	1	13

* From the Department of Surgery, Wayne University College of Medicine, Detroit, Michigan.
† The study is considered on total patient load rather than individual side of wounding.

TABLE III

Complications	Civilian Life			Military Service		
	Right	Left	Bilateral	Right	Left	Bilateral
Organizing hemothoraces (infected)	0	0	..	19	5	2
Organizing hemothoraces (uninfected)	1	0	..	8	0	0
Subphrenic abscess	0	0	..	6	1	..
Peritonitis	..	..	1	..	2	..
Biliary fistulas (thoracic)	..	..	0	3	0	..
Biliary fistulas (abdominal)	..	..	1	4	0	..
Fistulas of large bowel	..	..	0	..	1	..
Fistulas of small bowel	..	..	0	..	1	..

TABLE IV
RESULTS OF TREATMENT

Results	Civilian Life	Military Service
Deaths	1	2
Primary recovery	24	62
Delayed recovery	5	48

RESULTS

It is of interest that when a patient has been adequately cared for immediately after wounding, the late fatality rate is indeed relatively low. It then becomes a matter of dealing with the complications which occur. Of the deaths which occurred, one civilian died of fulminating peritonitis. Of the two deaths in military service, one person died of a fatal pulmonary embolus on the ninth postoperative day. This soldier had a perfect result from the post-wounding surgery, but apparently an unobserved thrombophlebitis had developed which was the cause of his death. The other death in the military group was the result of severe sepsis and multiple fistulas of the large and small bowel.

There were thirty-four severe chest complications in 112 wounded soldiers. There was only one such instance in the thirty wounded civilians. This is understandable when the missile of wounding is considered. Wounding in war is, as a rule, due to larger rifle missiles or to shell casing, whereas those in civilian life are the result of stabbing or gunfire of small caliber. Because of the size and nature of the wounding missile in war, clothing and other debris are often carried into the thoracic cavity.

It will be seen by referring to Table III that the problem of empyema has been placed under the heading of organizing hemothoraces. This was done because in the majority of instances this was the case. This approach also has been taken because a number of the so-called pure cases of empyema were treated in the same manner, namely, by decortication.

There were nine uninfected organizing hemothoraces. These were all successfully treated by decortication. There were twenty-six cases of infected hemothorax or empyema. Twelve of these were treated with pulmonary decortication; eight were successful and four could be considered failures or poor results, although it seemed likely that no patient would need further surgery.

In nine patients the intrapleural collection of purulent material was treated by rib resection and drainage. This was chosen for either of two reasons: it was thought because of the extreme illness of the patient that this was a more conservative method of approach, or the pocket was small and therefore did not justify a more formal procedure. In eight of the nine patients so treated it appeared that in only one instance would further surgery be needed.

In five patients as a result of disruption of the primarily closed chest wall wound no drainage was required, as the wound was adequately open on our first contact with them. These individuals all required further surgery to close the defect. In two this was done in the theater of operations. The remainder of the patients were sent to the zone of the interior.

No subphrenic abscess developed in the civilian group. There were seven such complications in the wounded soldiers. Of these, six occurred on the right side and one on the

left. All patients were treated with adequate drainage and recovered.

There were three instances of peritonitis, one occurring in the civilian group and two in the military group. Of the total, two died.

There were three instances of biliary fistula, (thoracic), all of which occurred in wounded soldiers. Two soldiers had a pure fistula which passed through the diaphragm into the pleural space and there formed a small pocket. It was possible in both these patients to aspirate pure bile which was uninfected. These patients were both treated with thoracotomy and decortication of the overlying lung. With re-expansion of the lung both fistulas closed. The third patient had empyema in association with biliary fistula. It was impossible to decorticate the lung, and a Schede type of thoracoplasty was done. When last seen the fistula had closed and it appeared that the outcome would be satisfactory. There were five instances of abdominal biliary fistulas in which it appeared that there had been injury to either the common or the cystic duct. The soldiers were sent to the zone of interior, and we have no record of their outcome.

It will be noted again by referring to Table III that the greater number of thoracic complications occurred in the wounds of the right side. Nineteen infected hemothoraces occurred in right-sided wounds, whereas in only five patients did this condition occur on the left. Even in those patients with supposedly uninfected hemothoraces all wounds were of the right side.

COMMENTS

The primary function of this paper is to attempt to relate what happens to the soldier with a thoracoabdominal wound after he leaves a forward station. The main bulk of the data has been obtained from the records of wounded soldiers, and those things which have happened to civilians under similar circumstances are really not of great importance to the paper. This is relatively true because, as has been previously mentioned, civilian wounding is not as a rule as severe as military wounding. In summary, we believe that there are certain pertinent facts which should be stressed. In the early publications on this subject after World War II, all of which dealt with the early treatment of thoracoabdominal wounding, it was evident that the left-sided wound was the more severe. This was primarily because of the severe bleeding which occurred from injury to the spleen. Of the left-sided wounds, complications occurred in only ten of 112 patients. In this group the spleen was injured in twenty-four instances. The principle group of complications occurred in those individuals in which the wound was on the right side and the liver had been injured.

There are further salient points to be collected from this study. In 112 military wounds the diaphragm was repaired, and both the chest and the abdomen were explored in 103 patients. In nine individuals in whom this was not done, complications occurred in all. Of the civilian group, twenty-two were explored and the diaphragm was closed. In the remaining group three hernias of the diaphragm developed which needed later repair.

We believe that this group of patients, especially the military, presents certain important points to be considered. There can be no doubt but what adequate immediate care of the patient who receives a thoracoabdominal wound is most important. Such treatment has reduced not only the immediate fatality in great amounts, but also the late fatality as is demonstrated herein. In this series only three of 142 patients died in the late period. Because we had opportunity to observe these patients for some time, it seems a certainty that the lasting effects should have been most minimal.

The results might have been in some instances much better. Much of this material was collected when the war was new to us. Certain mistakes were made and may be enumerated. Too often the wound below the diaphragm was not drained. This when not done on the right side resulted almost always in either thoracic or abdominal complications. In like manner, the incidence of pulmonary complication was increased when closed drainage of the chest was not done.

It thus seems that with good immediate care of the thoracoabdominal wound, which is the period of great life saving, the late complications which may be difficult can be cared for with a low fatality rate.

CONCLUSION

1. One hundred forty-two patients with thoracoabdominal wounds, who were observed after the fifth day of wounding, have been reported. In this group there were three deaths.

2. The treatment of these patients in the late period has been discussed.

Penetrating Wounds of the Neck

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OF all the topographic regions associated with injury in man, the area of the neck is most vulnerable to multiple anatomic and physiologic derangement. Injury from a bullet may result in a small penetrating cervical wound and still have traversed the trachea, the esophagus and a major blood vessel with few immediate clinical signs. Although the relative incidence of penetrating wounds of the neck is as frequent in civilian life as on the battlefield, diverse views regarding the need for early surgical exploration still prevail. The present study was undertaken in an effort to evaluate the need for early surgical exploration of all penetrating neck wounds as well as to correlate results obtained with the form of therapy employed.

The majority of reports dealing with wounds of the neck have been derived from the surgery of warfare. Only three neck wounds were reported from the Crimean War. Two of these were fatal and the third resulted in invalidism.[1] During the Civil War there were 4,114 cases of wounds of the neck with an approximate mortality of 15 per cent.[2] The relatively low mortality rate in that series may be attributed to the management of neck wounds during that period. The dangers of secondary hemorrhage resulting from the expectant therapy of vascular injury were well described. The importance of an adequate airway, wound débridement and wound drainage was a considered aspect of therapy.

From the Spanish American War 188 neck wounds were reported with a mortality of 18 per cent.[3] During World War I there were 594 cases reported with an approximate mortality of 11 per cent.[4] However, the great number of arterial aneurysms in the neck reported as delayed complications of neck wounds was evidence that expectant therapy was employed too often. The greatest number of deaths resulted from uncontrolled hemorrhage.

In the period between the two wars the literature was characterized by single case reports.[5–8] It is difficult to state what the general mortality in civilian neck wounds was during this period. An increasing number of arteriovenous aneurysms in the neck were reported and this fact may be interpreted as indicative of an increasing incidence of delayed exploration of neck wounds for vascular injury. Although great refinements in general surgical care were made up to the time of World War II, the advancements in the treatment of shock, infection and the metabolic aspects of injury were not well correlated with the technical aspects of penetrating neck injury. Although the errors of inadequate and delayed surgery had been well demonstrated in earlier years, sporadic reports of cases presenting the sequelae of surgical delay continued to appear.[9–11]

World War II afforded an impetus to more definitive expression of the surgical management of neck wounds. Tracheotomy appeared to have been employed at an earlier period; adequate drainage and control of infection for pharyngoesophageal wounds was instituted; and early therapy of vascular injury with attempts to restore continuity of the vessel was advocated. One series reported 851 cases of wounds of the neck with a mortality of 7 per cent.[12a] Lichtenstein[12b] reported a series of cases emphasizing the importance of anesthesia, early operation, adequate débridement and drainage. The need for early exploration of every cervical hematoma was strongly advised by Bailey.[13]

While combined organ injury in the neck does not permit standardization of surgical therapy, the importance of early exploratory cervicotomy cannot be overemphasized. Despite the advocation over 100 years ago of exploration of all wounds penetrating the platysma, bleeding neck wounds are still occasionally treated with pressure, expectant therapy and observation. Tracheal and esophageal injury occasionally is undiagnosed and results in death or serious complications. The following series of cases admitted to one county hospital is presented with the hope that it may

confirm the principle of obligatory surgery for penetrating neck injury.

MATERIAL FOR STUDY

This study consists of an analysis and review of 100 consecutive cases of penetrating wounds

TABLE 1
ANATOMIC DISTRIBUTION OF INJURIES (100 CASES)

Pharynx	8
Esophagus	1
Larynx	3
Trachea	10
Vascular	49
Brain and spinal cord	5
Brachial plexus	7
Thoracic duct	3
Total injuries	86

of the neck which were admitted to Parkland Memorial Hospital, Dallas, Texas, from January, 1948 through June, 1955. Injury was inflicted by bullet from a rifle or pistol in thirty-three cases, and by a shotgun in two cases. In sixty-five cases injuries were inflicted by stab or laceration with a knife or a similar instrument. The patients ranged in age from three to eighty-eight years, with the average age being 32.5 years. Eighty-three of the patients were males; seventeen were females.

All of the wounds penetrated through the platysma of the neck, or, if the point of entry was above the mandible or below the clavicle, traversed into the neck. In eighty-six instances injury involved the structure of the neck alone, while in fourteen cases there was associated injury to another part of the body.

Seventy-two cases resulted in uncomplicated recovery. In twenty-eight patients there were complications, resulting in eleven deaths. The anatomic distribution of injury is presented in Table 1.

DIAGNOSIS

The diagnosis of the severity and extent of injury which has occurred following a penetrating neck wound is often not evident. While obvious injury to the deep structures of the neck presents little problem as to the import or gravity of the wound, the benign appearance of a small entry wound is often beguiling of the true injury which lies beneath the platysma.

Various clinical signs have been used to diagnose the nature and extent of injury which has occurred following a penetrating wound of the neck. Bleeding and shock are often associated with vascular injury. Subcutaneous emphysema and airway obstruction may be indicative of tracheal laceration; and pain and dysphagia may indicate transection of the esophagus. However, these clinical signs may all be absent despite the presence of extensive injury.

In the present series of forty-four patients with vascular injury, 43 per cent demonstrated no evidence of peripheral vascular collapse and 70 per cent had no evidence of bleeding at the time of admission.

Of the thirteen patients who had obvious injury and penetration of the trachea and larynx, four failed to develop clinical signs sufficient to afford a diagnosis prior to surgical exploration.

The fact that only four patients of the eighty-three who were subjected to initial surgical exploration were without demonstrable injury at operation is further proof that clinical signs alone may be an erroneous guide for determining the nature of the injury. In other words, when an injury results in penetration of the platysma of the neck, the true extent of the damage can only be determined by early surgical exploration.

TREATMENT

The general principles of therapy for the management of penetrating wounds of the neck are (1) adequate treatment for shock, (2) provision for an adequate airway and the restoration of normal respiratory mechanics, (3) early exploratory cervicotomy with definitive surgery and (4) treatment of associated injuries.

The therapy for hemorrhagic shock is whole blood replacement. If hemorrhage is massive and continuous, it is preferable to administer O-negative blood rather than to chance life by awaiting a properly typed and cross matched specimen. Until the time when any blood is available, lactate-Ringer's solution may be used as a volume expander. Dextran in saline and plasma solutions are associated with too high an incidence of complications to warrant their use at present.

Immediate provision for an adequate airway is of prior importance. Tracheotomy should be performed on slight suspicion of impairment of airway. To wait until obvious gross clinical signs of airway obstruction due to fluid or gas in the neck appear is to participate in unnecessary delay. Mediastinotomy is likewise indi-

TABLE II

RELATION OF SURGICAL EXPLORATION TO MORTALITY AND MORBIDITY

Time of Surgical Exploration	Deaths	Complications	Morbidity (%)	No Complications	Bleeding on Admission	Shock on Admission
Within 6 hr. after injury (73 cases)	3 (4%)	9	12	61	17 (21%)	32 (39%)
Over 6 hr. after injury (10 cases)	2 (20%)	5	50	3		
No surgical exploration	6 (35%)	3	18	..	2 (11%)	6 (33%)

cated with only minimal mediastinal emphysema. Auscultation for a mediastinal crunch is often rewarding in revealing minimal amounts of air and affording an opportunity for optimal therapy.

The treatment of associated injuries may often take precedence over the neck injury. The high incidence of intrathoracic involvement in supraclavicular wounds should always lend suspicion to the presence of either a pneumothorax or hemothorax, or both. X-ray examination of the chest may be helpful. Thoracentesis and possible water seal drainage of the chest may be instituted and may aid in the quantitation of hemorrhage.

The importance of early exploratory cervicotomy in the present series of penetrating neck wounds is presented in Table II. Initial surgical exploration of wounds of the neck was performed in eighty-three cases with a mortality of 6 per cent (five deaths). In seventeen cases in which no surgical exploration was attempted there were six deaths yielding a mortality of 35 per cent. In addition, when cervicotomy was performed within six hours following injury the mortality rate was 4 per cent. When surgical exploration was deferred beyond six hours following injury the mortality rate rose to 20 per cent. The progressive increase in morbidity relative to the time of exploration is further presented in Table II.

The performance of definitive and adequate surgery in exploration of a neck wound is of great importance. Adequate visualization of the great vessels, the exploration of the missile track to its depth and the definitive treatment of vascular, tracheal, esophageal and nerve injury is desirable in every instance.

Although local anesthesia is adequate for superficial neck exploration, general anesthesia is necessary for the majority of exploratory cervicotomies. Agents such as pentothal® or cyclopropane which tend to stimulate somatic-visceral reflexes or reflexly affect pulmonary or cardiac function should be avoided. In addition, the maintenance of a high oxygen content to prevent anoxia and asphyxia in any degree is necessary. In the present series nitrous oxide-oxygen-ether was most frequently employed.

Tracheal intubation should be routine. It is a prerequisite in suspected tracheal injury and is obligatory if the pleura is penetrated by injury or surgery.

Premedication with atropine in adequate dosage to prevent reflex arrhythmia is desirable with operative manipulation about the neck. It is wise to withhold narcotics pending complete evaluation of the neck injury. Positioning of the patient upon the operating table is dependent upon the location of the injury and the anticipated incision. Surgical draping must allow for a generous incision or an extension of the incision if proximal control of a perforated vessel becomes necessary.

During induction and surgery the possibility of air embolism should be considered and open venous channels ligated without delay. The surgeon and anesthetist must be alert to any changes in the respiratory pattern incident to the delayed onset of pneumothorax.

Finally, the status of the vocal cords should be determined by direct visualization at the completion of operation.

VASCULAR INJURY

Injury to the vascular system within the neck occurred in forty-four cases in the present series. The mode of injury was by bullet in

thirty-three instances and by knife or a similar instrument in eleven cases. The anatomic distribution of vascular injuries is presented in Table III. There were six deaths among these cases, yielding a mortality rate of 14 per cent. The most common cause of death associated with vascular injury was hemorrhage. That delay of surgical exploration is not warranted is attested to by the data presented in Table IV. In those instances in which surgical exploration was performed within six hours following injury the mortality was 6 per cent; when surgical exploration was delayed beyond six hours following injury the mortality rate was 25 per cent; and when expectant, non-operative therapy was employed, the mortality was 100 per cent.

TABLE III
ANATOMIC DISTRIBUTION OF VASCULAR INJURIES
(FORTY-FOUR CASES)

Subclavian artery	4
Vertebral artery	2
Thyrocervical trunk	2
Common carotid artery	2
Internal carotid artery	1
External carotid artery	2
Superior thyroid artery	1
External maxillary artery	1
Transverse cervical artery	1
Ascending cervical artery	1
Facial artery	2
Innominate vein	3
Subclavian vein	4
Internal jugular vein	11
External jugular vein	9
Anterior jugular vein	3
Total injuries	49

While the surgical management of injuries involving many minor arteries and veins in the neck is direct and simple, injuries to the subclavian artery, common or internal carotid arteries, the vertebral artery and major veins present special problems in therapy, and because of their importance they are dealt with separately.

Common or Internal Carotid Artery. Injury to the common carotid artery occurred in three cases. Two injuries resulted from bullet penetration and one occurred as a result of knife stabbing. There were two deaths with a mortality of 67 per cent.

One patient (bullet wound) was subjected to immediate surgical exploration with ligation of the common carotid and uneventful recovery. The second patient (stab) was not bleeding upon admission and was expectantly treated until massive hemorrhage ensued. A clamp was placed on the internal carotid artery with subsequent ligation. Shortly after this emergency measure the patient became comatose. No complete surgical exploration was performed. The wound continued to bleed slowly and the patient's condition deteriorated. He remained comatose until death on the fourth day following injury. The following case (bullet wound) illustrates the major complications of vascular injury when expectant therapy and delayed surgery have been employed.

TABLE IV
VASCULAR INJURIES—RELATION OF SURGICAL EXPLORATION TO MORTALITY

Type of Injury	No. of Cases	No of Deaths	Mortality (%)
Surgery Performed within Six Hours			
Stab wound	27	1	6
Gunshot wound	7	1	
Surgery Performed after Six Hours			
Stab wound	5	1	25
Gunshot wound	3	1	
No Surgery Performed			
Stab wound	1	1	100
Gunshot wound	1	1	

CASE I. A twenty-two year old Negro man sustained a bullet wound with point of entry at the base of the left neck immediately posterior to the sternocleidomastoid muscle. No exit wound was present. There was no active bleeding and the patient was not in shock at the time of admission. X-rays located the bullet in the mid-lateral base of the right neck.

One hour after admission tracheotomy was performed because of diffuse neck swelling. In performing this procedure a perforation of the trachea was discovered. No further exploration was attempted. The following day there was evidence of an arteriovenous fistula at the base of the right neck. In addition, the patient's course was complicated by fever and hemorrhage in small quantities from the tracheotomy wound. His course improved, however, and upon that basis he was discharged from the hospital. In one month he was

to return for surgical repair of the auriculoventricular aneurysm.

On the forty-fifth day after injury the carotid-jugular arteriovenous fistula was excised with restoration of continuity of the carotid artery. Heparin was employed postoperatively.

On the third postoperative day a wound hematoma was evacuated. On the twelfth postoperative day wound bleeding occurred. Operation was performed and hemorrhage from the carotid artery was controlled by pressure. Bleeding ceased and the patient was returned to his room. The following day bleeding resumed and surgery was performed with ligation of the carotid artery and jugular vein. The patient was in a critical condition. Ten days following this procedure bleeding resumed. Thoracotomy was performed and revealed massive infection with necrosis of the carotid artery. The subclavian and innominate arteries were ligated. Hemorrhage resumed shortly after this procedure and the patient died seventy-eight days after injury. Examination revealed necrosis of the innominate artery at the site of ligation.

Definitive early surgery is the most conservative form of therapy. Expectant therapy is radical in that it results in an inordinately high mortality rate.

The mortality and complications which result from injuries to the common and internal carotid artery are dependent upon the type of wound, period of shock endured, age of the patient and the management of the injured vessel. Adequate exposure of the vessel and associated injury is necessary in every instance. While emergency clamping of a vessel may be a heroic procedure that may save life, it is better to control bleeding with a finger or artery clamp until the true extent of damage is determined by adequate exposure.

The correction of continuity of the carotid artery should always be considered. Knife wounds or lacerations usually lend themselves to direct suture of the vessel after débridement. Adequate length is generally available. However, gunshot injury of the carotid artery presents difficulty because of the associated effect of blast injury upon the arterial wall. Moore et al.[14] have studied contiguous arterial damage resulting from bullet wounds and have demonstrated that intimal damage always extends beyond the area of the damage estimated by gross inspection. These authors advocated resection of the vessel at least 3 mm. beyond the area of gross external damage and axial anastomosis. Failure to recognize the extent of blast injury which has resulted from a bullet wound to a major vessel leads to inadequate débridement and a high incidence of anastomotic breakdown with secondary hemorrhage.

In those instances in which considerable shortening of the vessel has resulted due to injury and débridement, a graft or prosthesis may be employed. However, the decision to maintain continuity of the vessel should be guided by the age of the patient and the extent of injury. In cases in which there is a great deal of necrosis of tissue attending a bullet wound or other severe injury, it may be judicious to ligate both ends of the severed carotid artery rather than attempt restoration of continuity. In other words, the risk of cerebral complications secondary to ligation may be of lesser importance than the risk of death from secondary infection and hemorrhage from anastomotic breakdown.

Experience with carotid artery ligation has shown that the morbidity and mortality attending ligation in the young is low, and increases in the adult and more particularly in the older age groups.[15] The cerebral complications are often related to ascending thrombosis rather than initial ischemia.[16] Regional heparinization following anastomosis or repair does not appear to be justified in view of the complications incident to its use and the questionable benefit obtained.[17] Concomitant ligation of the internal jugular vein has not resulted in any reduction in cerebral complications.[18] The avoidance of prolonged shock and the early performance of definitive surgery with possible restoration of continuity appears to be the only method available to effect a consistent reduction in mortality and morbidity following carotid artery injury.

Subclavian Artery. Injury to the subclavian artery occurred in four cases. Three were due to a high velocity missile and one was caused by a knife wound. There was one death. All four patients were admitted to the hospital in shock and none of the wounds was actively bleeding. Two patients underwent immediate surgical exploration with proximal and distal ligation of the vessel about the perforation. One patient had an uneventful recovery; in the other a wound abscess developed necessitating drainage. The remaining two patients were treated expectantly with transfusions, and observed. Both were finally subjected to de-

layed surgical exploration by thoracotomy. One was operated upon on the twenty-sixth day following injury with successful removal of a false aneurysm of the subclavian artery and ligation of the vessel. The following case resulted in a fatality and serves to demonstrate the lethal dangers involved in expectant therapy.

CASE II. A thirty-three year old Negro man sustained a bullet wound with point of entrance overlying the left sternoclavicular joint. The point of exit was located immediately to the left of the posterior midline at the level of T3. The patient was in a state of severe hemorrhagic shock on admission. He was treated expectantly with whole blood replacement and responded satisfactorily. X-ray, however, showed a persistent homogenous density in the left upper lung field and mediastinum.

On the eighth day after injury thoracotomy was performed through the left second interspace. Upon separation of the lung substance from the mediastinum, exsanguinating hemorrhage occurred. Postmortem study revealed that the hemorrhage had been the result of earlier injury to the left subclavian artery and innominate vein.

The surgical management of subclavian artery injury essentially consists of early definitive surgery. Injury should especially be suspected when there has been an episode of unexplained shock with the route of penetration being directed posterior to the clavicle. Hemorrhage may be completely intrathoracic so that immediate closed water seal drainage of the pleural cavity should be instituted when hemothorax is present in association with possible subclavian artery injury.

Opinion varies as to the surgical approach to the subclavian vessels. In the present series the superior approach with resection of a portion of the clavicle, as advocated by Elkin,[19] was employed in the two cases of early exploration. This approach appears to afford more direct exposure of the vessels for repair and ligation. When hemorrhage is profuse, rapid intercostal thoracotomy may be performed to effect proximal control of hemorrhage, followed by more leisurely exploration from within the chest and via the neck.

As with other major vessels, suture and repair following adequate débridement should be attempted. However, in cases of extensive laceration or bullet wound one need not hesitate to perform proximal and distal ligation of the damaged vessel. The incidence of gangrene in the upper extremity is low[20] and ischemic symptoms in the upper extremity following ligation occur infrequently and have not developed in any of the patients in our series to date.

Vertebral Artery. There were two instances of injury to the vertebral artery in the present series. Both of these were inflicted by bullet wounds and one resulted in death.

There are few injuries involving major vessels so constantly complicated by as high a mortality rate as that following injury to the vertebral artery. Clinically one should suspect vertebral artery injury when one of the following is present: (1) hemorrhage from a posterior or lateral neck wound not controlled by proximal occlusion of the common carotid artery, and (2) bleeding from the posterior lateral neck area in association with the fracture of a cervical transverse process.

Injuries of the extraosseous portion and the intravertebral portions of the vertebral artery are most resistant to measures for the control of hemorrhage. In addition, retrograde flow from intracerebral anastomotic channels further complicates simple proximal ligation. A number of comprehensive and excellent case reports concerning injury to the vertebral artery have appeared in the literature. Matas[21] reviewed twenty-two cases of wounds of the vertebral artery of which nineteen died. Case presentations and reviews by Perrig,[22] Heifetz[23] and Elkin[24] have stressed the pertinent anatomic and clinical features of wounds of the vertebral artery. The mortality of cases in the literature has generally exceeded 50 per cent.

Injury to the first or extraosseous portion of the vertebral artery can occasionally be dealt with by proximal and distal ligation. Hemorrhage due to injury to the intraosseous portion of the vessel is extremely difficult to control. Successful ligation of the intraosseous portion of the vertebral artery has been performed in relatively few instances. Identification of the vertebral artery in the normal individual is difficult; identification when there is associated hematoma and distorted anatomic relationships may be virtually impossible. The technical difficulties which the surgeon encounters when attempting to ligate the vertebral artery at the base of the neck have been stressed by Matas. In more than half of the cases in one series the common carotid artery or the thyrocervical trunk was mistaken for the vertebral

artery and inadvertently ligated. Since many of the aforementioned difficulties are often insurmountable, proximal control of the vertebral artery should be effected by subclavian artery ligation. The high mortality due to uncontrolled hemorrhage from the vertebral arteries indicates that delay or secondary measures employed to control bleeding have generally been ineffective. While retrograde flow of the distal cut portion may result in additional bleeding, it usually is minimal.

The following case is illustrative of a few of the pitfalls surrounding the diagnosis and therapy of vertebral artery injury.

CASE III. A twenty-seven year old white man sustained a bullet wound with point of entry over the left maxilla and point of exit in the left posterior neck at the level of C2. The patient was in a state of peripheral vascular collapse and there was active bleeding from the exit wound on admission to the hospital. Hemorrhage ceased spontaneously shortly after admission. X-rays failed to reveal a fracture but the left transverse process of the second cervical vertebra was not well visualized. Whole blood replacement was begun.

Seven hours after injury an episode of massive bleeding occurred. The patient was taken to the operating room where tracheotomy was performed and active hemorrhage was controlled by occlusion of the left common carotid artery. Left common carotid ligation followed. The bullet tract was not explored because it was thought to traverse a technically inaccessible area.

In the recovery room following surgery active bleeding from the exit wound again occurred. The patient was returned to the operating room, the posterior wound was opened widely and the fractured transverse process of the second cervical vertebra was palpated. It was determined that hemorrhage was due to a severed left vertebral artery. The medial half of the left clavicle then was resected and the first portion of the left subclavian artery was ligated in continuity. Drainage was established through the open exit wound. No further bleeding from the posterior wound ensued. Other than minor hemorrhage from a small branch of the external carotid artery, which was controlled by ligation, recovery was uneventful.

Injuries to Major Veins. There were eighteen injuries to major veins in the neck in the series, distributed as follows: innominate, three cases; subclavian, four cases; and internal jugular, eleven cases. There were four deaths associated with major vein injury.

The surgical management of injuries to veins consist of early recognition and exploration and adequate definitive surgery. It is best to effect proximal and distal ligation of all veins of the neck except in the single instance when both internal jugular veins have been transected. Under these circumstances, because of the cerebral complications resulting from bilateral venous occlusion, an attempt should be made to restore the continuity of one of the internal jugular veins.[25]

TABLE V
INJURIES TO TRACHEA AND LARYNX

Case No.	Type of Injury	Treatment	Remarks
5	Gunshot wound (L)*	None (unrecognized injury)	Death
7	Gunshot wound (L)*	Delayed tracheotomy	Death
31	Gunshot wound	Tracheotomy and mediastinotomy	Uncomplicated
35	Gunshot wound	Tracheotomy	Uncomplicated
43	Gunshot wound	Tracheotomy	Death (vascular injury)
64	Gunshot wound	Tracheotomy	Uncomplicated
93	Gunshot wound	Tracheotomy	Associated brain injury
1	Stab	Suture and drainage	Uncomplicated
49	Stab	Suture and drainage	Uncomplicated
58	Stab	Tracheotomy	Uncomplicated
69	Stab	Tracheotomy	Uncomplicated
75	Stab (L)*	Tracheotomy and mediastinotomy	Uncomplicated
92	Stab	Tracheotomy	Uncomplicated

* Injury involving larynx.

INJURY TO THE LARYNX AND TRACHEA

In the present series thirteen patients suffered from injury to the air passages. Of these, three died. Six injuries resulted from knife stabbing and seven were produced by bullet penetration. The data are presented in Table v.

Two deaths were directly due to complications resulting from failure to recognize the extent of injury to the trachea and larynx. The third death was due to secondary hemorrhage from an associated vascular injury. Therapy in those patients subjected to surgery consisted of tracheotomy alone in seven cases, tracheotomy and mediastinotomy in two patients, and primary suture and drainage of small tracheal lacerations in two instances. In eleven cases subjected to early surgery the course was uncomplicated in respect to tracheal or laryngeal injury.

Therapy for injuries of the trachea and larynx include the following principles:

1. Early recognition of air passage injury including a high index of suspicion in wounds at or near the midline. Early recognition, however, may be difficult since signs of injury are frequently absent. Signs of airway obstruction may not be present if edema is absent or insufficient to compromise the air passages. Subcutaneous emphysema may not occur if there is a large open external wound. Hemoptysis requires bleeding into the air passage for its production.

2. Provision for an adequate airway. Tracheotomy is desirable upon minimal evidence of impairment of airway. The early clinical signs—an increased respiratory rate, a slight expiratory grunt, an auscultatory mediastinal crunch—should lead one to decide early in favor of tracheotomy. The ill effects of prolonged respiratory obstruction atelectasis, pulmonary edema and congestion are often irreversible phenomena and may result in death. Post-tracheotomy care of the tracheobronchial tree including adequate wetting agents and bronchodilators are a great adjunct in the maintenance of normal pulmonary physiology.

3. Definitive surgery. Tracheotomy alone, while often life-saving in providing the needed airway, may be inadequate if further exploration of the damaged trachea is not performed. Adequate determination of the defect in the airway should be performed with débridement of dead tissue. Closure of a defect by primary suture, graft or use of the overlying tissues may be effected. In fractures of the thyroid and simple lacerations of the trachea, closure and tracheotomy are all that is necessary. The difficulties of closure arise with large defects. Maisel and Dingwall[26] have found from experiments on dogs that primary suture of the completely divided trachea can be performed without difficulty, and they indicate that resections are feasible if the cut ends can be mobilized. Patients with injury involving the integrity of the tracheal rings or cricoid are candidates for skin and cartilage grafts as advocated by Lynch.[27] Burton and Anderson[28] proposed split-skin graft over a stent of rubber tubing with later placement of an acrylic obturator in extensive tracheal defects. Many authors have used fascial grafts. Morfit et al.[29] have satisfactorily bridged defects of the trachea with a permanent prosthesis of polyethylene tubing.

4. Drainage. In view of the open route into the superior mediastinum through which accumulations of air or infected material can travel, drainage by mediastinotomy is often necessary in wounds of the area. Extensive mediastinal emphysema should be treated with immediate mediastinotomy in addition to tracheotomy.

TABLE VI

INJURIES TO THE PHARYNX AND ESOPHAGUS

Case No.	Type of Injury	Treatment	Remarks
5	Gunshot wound (E)*	No therapy	Lesion unrecognized; death
11	Gunshot wound	None	Uncomplicated
14	Gunshot wound	Drainage only	Uncomplicated
91	Gunshot wound	Suture and drainage	Uncomplicated
93	Gunshot wound	Drainage only	Uncomplicated
2	Stab	Drainage only	Uncomplicated
13	Stab	Suture only	Wound abscess
25	Stab	None	Recurrent hemorrhage
75	Stab	Suture and drainage	Uncomplicated

* Esophagus; all other injuries are to pharynx.

Other complications must be dealt with as they are met. With the forced respiration of airway obstruction, acute gastric dilatation is a common finding in association with tracheal injury, and requires nasogastric suction. Aspiration of blood or debris at the site of injury may occur and will necessitate careful attention to the toilet of the tracheobronchial tree, possibly bronchoscopy. Infection may be controlled with antibiotics. Stricture formation is a late complication of extensive laryngotracheal injury and may require repeated dilatations or reconstructive surgery.

INJURY TO THE PHARYNX AND ESOPHAGUS

There were nine cases of injury to the pharynx and esophagus in the present study. Seven patients had uneventful recoveries and major complications developed in two, one of whom died. The data is presented in Table VI. It is significant that the latter three patients were treated expectantly without drainage.

The principles of therapy for wounds of the pharynx and esophagus include early recognition and surgical exploration with definitive surgery plus provision for adequate drainage.

Dysphagia may be present. The presence of blood in the pharynx or stomach in association with a penetrating neck wound may be indica-

tive of food passage injury. Accumulations of fluid or gas within the cervical mediastinum may be present. Occult injury may occasionally be diagnosed with the aid of esophagoscopy or the instillation of radiopaque material into the pharynx and esophagus.

Early definitive surgery is necessary to treat many of the severe complications which follow perforation of the pharynx or cervical esophagus. Because of the location of the esophagus and pharynx deep within the neck, the probability of associated injury is great, and may bear serious consideration relative to the technical management of closure and repair. Simple lacerations and small defects of the pharynx and esophagus can be sutured. If a small perforation of the pharynx is inaccessible to suture, satisfactory healing may occur with drainage alone. Destructive injuries of the esophagus and pharynx require more extensive surgical procedures, including débridement and repair of the defect. Primary anastomosis of the severed cervical esophagus is now an acceptable procedure with end-to-end anastomosis. Primary reconstruction may be considered for large defects. Split-thickness skin grafts,[30] fascia lata grafts[31] and preserved aorta bridges have been utilized with success for the restoration of continuity of the pharynx and esophagus.

Drainage of perforating wounds of the pharynx and esophagus is desirable as a routine measure. Secretion accumulation in the subcutaneous tissues, abscess formations and mediastinitis can largely be prevented if adequate wound drainage is effected. Persistent fistula formation and strictures of the esophagus may result from extensive injury and often require secondary surgery.

Nasogastric intubation is important for early decompression and as a route for feeding. Spillage of contaminated material at the site of the wound can be prevented by the elimination of vomiting and oral intake. Antibiotic administration is a necessary adjunct to therapy.

NERVE INJURY

Injury to the brachial plexus occurred in seven cases in the present series. In four cases there was severance of nerve fibers, two of which were treated by primary suture followed by slow but progressive improvement in neurologic function. The remaining two patients with severance of nerve fibers did not undergo primary nerve repair and their present status is not known. Concussional injury to the brachial plexus by bullet wound occurred in three cases. Full return of function occurred in each case; physiotherapy was used routinely.

Penetrating wounds of the neck may produce nerve injury either by direct severance of fibers or by the concussional effect of high velocity missiles. Early exploration of neck wounds offers an opportunity for prompt surgical repair and restoration of nerve continuity. Davis et al.[32] believe that immediate end-to-end apposition of the severed nerve segments following débridement is desirable and provides optimal conditions for growth and repair. Following nerve injury, the employment of physiotherapy should be instituted until maximal return of function has been obtained.

Injuries to the vagus, phrenic and recurrent laryngeal nerves occasionally occur following a penetrating injury to the neck. Transection of the vagus nerve is important not only in the resulting cord paralysis but also because of the changes in pulmonary function. Klassen et al.[33] have shown that cervical vagal section abolishes the cough reflex of the homolateral bronchial tree. The significance of this fact in the maintenance of adequate tracheobronchial toilet is apparent. Injury to the phrenic nerve may not produce paralysis of the hemidiaphragm because of the presence of an accessory phrenic nerve in 75 per cent of subjects as studied by Kelley.[34] Whenever possible, severed vagus, phrenic and recurrent laryngeal nerves should be sutured. In the present series no known injuries to any of these nerves followed neck wounding.

THORACIC DUCT

Injuries to the thoracic duct occurred in three cases. In one case the duct was repaired with the complication of chylous external drainage for several days postoperatively. In two patients the duct was ligated without complication. The importance of injury to the thoracic duct lies in the appreciation of the complications which follow as well as in the recognition and therapy of the injury. Chylothorax may follow, and does not require the penetration of the pleura for its production. In past years chylothorax has carried a substantial mortality.[35] Lymphatic fistula may be a distressing sequela. The thoracic duct may be ligated without complication and this cir-

cumvents the development of chylothorax or fistula.[36] Repair may be performed, but is often associated with a high incidence of breakdown.

COMPLICATIONS

Seventy-two cases in the present study had uncomplicated recoveries. Of the remaining twenty-eight cases, there were thirty complications and eleven deaths. The most frequent complications were secondary hemorrhage, sepsis and pulmonary dysfunction. The data are presented in Table VII.

TABLE VII
COMPLICATIONS OF PENETRATING WOUNDS OF THE NECK*

Infection:	
Wound infection	3
Mediastinitis	1
Sepsis without localization	2 (1 death)
Secondary hemorrhage	8 (5 deaths)
Arteriovenous fistula	1 (death)
Lymphorrhea	1
Cardiac arrest	1 (death)
Central nervous system:	
Brain injury	3 (1 death)
Cord injury	2 (2 deaths)
Pulmonary:	
Atelectasis	2
Hemothorax	4 (1 death)
Pulmonary obstruction	1 (death)
Pulmonary failure	1 (death)

* Thirty complications in twenty-eight cases (eleven deaths).

Secondary hemorrhage occurred in eight patients and an arteriovenous fistula developed in another. There was a mortality rate of 67 per cent for those patients in whom secondary hemorrhage developed. The high mortality in this group is probably preventable if adequate and definitive surgical exploration is performed for all penetrating neck wounds.

Wound abscesses developed in four patients; wound drainage was not effected at the time of surgery in two of these cases. The universal contamination of penetrating wounds of the neck, the associated tissue destruction with high velocity missiles, and the frequent spillage of alimentary secretions dictates the almost routine use of antibiotics and wound drainage. In two cases sepsis without localization continued for a prolonged period following neck injury.

Pulmonary complications were present in eight patients. In four instances hemothorax developed following missile penetration into the thorax. Closed water seal drainage was employed in all four instances with gradual resolution and clearing of the pleural space. The remaining four pulmonary complications were probably the result of prolonged respiratory obstruction. Atelectasis was present in two cases, irreversible pulmonary failure in one case and pulmonary obstruction in one case.

Four of the five neurologic complications resulted from brain or cord damage at the time of the original injury. In the remaining case, however, coma and death followed carotid ligation.

DEATHS

There were eleven deaths in the series of 100 patients studied, eight inflicted by bullets and three resulting from knife stabs. A summary of the pertinent clinical features of fatal cases is presented in Table VIII. Neck injury alone was responsible for the death of six patients. In this group there were four cases of vascular injury alone, one patient with laryngeal injury alone, and one instance of combined tracheal and vascular injury.

Five patients had associated injury in another portion of the body. Cord and brain injury were the most frequent sites of additional injury.

While the cause of death cannot always be ascertained, a positive correlation exists in the present series between death and surgical exploration. Early surgery was attempted in only one patient of the eleven who died. In every death due to neck injury, with this single exception, there was either no operation, delay in operation or inadequate exploratory operation. It is possible that fatality was unavoidable in the five cases in which there was associated additional injury. Death in the one instance of attempted early surgery was due to cardiac arrest. In the remaining five cases it is possible that death could have been prevented by early adequate therapy.

SUMMARY

An analysis and review of 100 penetrating wounds of the neck in a single civilian hospital has been presented.

The mortality in the series was 11 per cent, or approximately the same incidence of death reported for neck wounds from all of the great wars beginning with the Civil War. Civilian

TABLE VIII

SUMMARY OF DEATHS DUE TO PENETRATING WOUNDS OF THE NECK

Patient	Age (yr.), Race and Sex	Type of Injury	Neck Injury	Time of Death	Other Injury	Remarks
L. L.	19, N, M	Gunshot wound	Perforation of esophagus and larynx	11 hr.	Transection of cord C6	No surgery performed: hematemesis, pulmonary obstruction; death; autopsy performed
A. M.	52, W, M	Gunshot wound	Perforation of larynx; diffuse thyroid hemorrhage	36 hr.	Penetration of abdomen (negative exploration), fractured mandible	Neck not explored: laparotomy and delayed tracheotomy; shock, pulmonary dysfunction, Cheyne-Stokes respiration; death; autopsy performed
E. M.	25, N, M	Gunshot wound	Hematoma in neck	2½ hr.	Subarachnoid hemorrhage, laceration of brain	No surgery performed: patient comatose; Cheyne-Stokes respiration; death; autopsy performed
C. B.	65, W, M	Laceration	External carotid artery, external jugular vein	13 days	Fractured skull, retroperitoneal hemorrhage, subarachnoid hemorrhage, cerebellar hemorrhage and fractured pelvis	Shock, then restoration; cardiac failure, central nervous system signs; sudden death; autopsy performed
E. J.	38, N, F	Gunshot wound	Penetrating wound; no apparent injury	43 days	Transection of cord C7, quadriplegia	Apparently no complications from neck injury (other than neurologic); sepsis, hypotension; death; no autopsy performed
J. T.	23, N, M	Stab	Subclavian artery, vertebral artery	8 days	None	Inadequate exploration: massive hemorrhage 104 hr. after admission; branch of subclavian artery ligated; massive hemorrhage; death; autopsy performed
A. V.	35, N, M	Stab	Internal carotid	13 days	None	Inadequate exploration: admitted to emergency room; pressure stopped bleeding; patient transfused; recurrent hemorrhage and shock; vessel ligated in emergency room; Cheyne-Stokes respiration; wound bled for 4 days; tracheotomy; death; no autopsy performed
C. D.	29, N, F	Gunshot wound	Penetrating wound, injury unknown	17 days	Cord injury to C7 with paraplegia; left hemothorax; penetrating wound to abdomen (exploration negative)	Neck not explored: paraplegia; fever, anemia, neck swelling, hemothorax; death; no autopsy performed
C. M.	22, N, M	Gunshot wound	Trachea, right common carotid, right jugular vein	50 days	None	Inadequate and delayed exploration; 1st admission—tracheotomy (no neck exploration); 1st postoperative day: bruit, base of neck; arteriovenous fistula. 2nd admission 4 wk. later—arteriovenous fistula (carotid artery and jugular vein repaired); 3rd postoperative day: hematoma; wound evacuated. 12th postoperative day: hemorrhage and shock; operation; pressure applied to site. 14th postoperative day: at operation, carotid artery and jugular vein ligated; patient critical. 24th postoperative day: hemorrhage; thoracotomy performed; subclavian and inominate arteries ligated. 33rd postoperative day; hemorrhage and death; autopsy performed
E. S.	33, N, M	Gunshot wound	Subclavian artery, innominate vein	8 days	None	Delayed surgery; observed for 8 days; thoracotomy performed; massive hemorrhage after lung separated from mediastinum; immediate death
H. C.	48, N, M	Gunshot wound	Left subclavian vein	5 hr.	Left hemothorax	Thoracotomy; cardiac arrest during operation; death

neck injuries, both in respect to the extent of damage and the time of therapy, are of a favorable type compared to war wounds. Yet the mortality rate in the present series fails to indicate that any comparative reduction in mortality has been obtained in the past 100 years, despite the greater amenability of civilian wounds to early definitive surgery. Every death in the present series, other than those due to associated injury, may be shown to be due to either an inordinate delay in surgery or inadequacy of surgical exploration. The devious paths which penetrating missiles into the neck may take, the variability of hemorrhage or associated signs seem only to offer the surgeon a false sense of security as to the true extent of damage in the neck. For these reasons it appears safe to conclude that the only certain method of determining the true extent of injury beneath the platysma is by early exploratory cervicotomy. In this respect the platysma in the neck may be compared to peritoneum of the abdomen in that the extent of injury when there is penetration of the latter can only be determined by exploratory laparotomy. This thesis is further attested to by the fact that there were only four instances of negative exploration in the eighty-three cases which were subjected to exploratory cervicotomy. In addition the mortality rate in those instances where exploration was performed was 6 per cent, compared to a death rate of 35 per cent when surgery was deferred or omitted.

The principles of therapy for the management of penetrating neck wounds consist of adequate preoperative resuscitative measures, followed by immediate surgical exploration and definitive surgery. Whole blood transfusions, provision for an adequate airway and the emergency control of hemorrhage are imperative.

While a few minor wounds in cooperative patients may be explored under local anesthesia, general anesthesia with tracheal intubation is a prerequisite for surgical exploration. The wide surgical exposure necessary in many instances plus the reflex complications attending manipulations about the neck require obtundation of visceral reflexes by adequate atropinization and general anesthesia. In the present series local anesthesia was employed in twenty instances. The remainder of the cases were anesthetized with gas-oxygen-ether via endotracheal tube or tracheotomy.

The most common complications in the series were hemorrhage, sepsis or pulmonary dysfunction.

These complications may be reduced to a minimum by early definitive surgery, provision for an adequate airway, the restoration of normal respiratory mechanics, and adequate wound débridement and drainage.

Injuries to the carotid, subclavian and vertebral arteries deserve special consideration in therapy. Lacerations of major vessels by a knife or sharp instrument may often be treated by repair or axial anastomosis. Gunshot injuries, however, require wide débridement of the injured vessel which often results in inadequate length for anastomosis. While the restoration of continuity by prosthesis under these circumstances is desirable, it is not always mandatory and may be unwise in the presence of extensive contiguous injury.

The ill effects of prolonged respiratory obstruction, even if relieved by delayed tracheotomy, are often irreversible. Asphyxia, pulmonary edema and atelectasis are preventable complications if tracheotomy is performed on slight indication of impairment of airway. The maintenance of adequate tracheobronchial toilet following tracheotomy is an important adjunct to therapy.

The incidence of sepsis and wound infection is correlated with the type and extent of injury. Shotgun injury may be associated with extensive destruction of skin and much tissue injury while single missile bullets result in a comparative lesser degree of necrosis. Knife wounds usually result in little or no necrosis of tissue. Adequate débridement of all devitalized fragments including vascular, esophageal and tracheal tissues should be performed. Wound drainage is desirable as a routine measure. Antibiotics should be employed in all instances except in cases with minimal injury due to a clean knife wound.

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DISCUSSION OF PAPERS BY DRS. SHEESLEY, HEYDINGER AND ROETTIG, AND FOGELMAN AND STEWART

Charles G. Johnston (Detroit, Mich.): I wanted to discuss these two papers because they bring up some very fundamental points of view.

I believe the latter paper might be extended to deal with stab wounds or injuries over areas where any large and important vessels are especially concerned. Of course, in the neck there are other structures to worry about aside from large vessels.

It has been my experience that the average physician who treats such cases has a notion that he can depend on signs and symptoms of blood loss (by determination of swelling or fast pulse or by other means) in deciding the extent of injury.

This belief is certainly not so. We have seen patients with large, transected vessels in whom bleeding could not be determined on physical examination. Therefore, we have insisted that any injury which *might* be near a vessel of any size or extent which could be seriously traumatized must be explored whether it is in the neck, the groin or the axilla.

These injuries must be explored early because

delay may cause the blood to form a small hematoma. Some hematomas will be absorbed; others will leak. If the injuries are ignored for a few days, not only will the operative field be difficult but also the patient will be partially depleted and less viable.

In relation to the chest problem, I wish I could conclude that all these patients should be operated upon. I am sure that in this respect individualization is exceedingly important. We have tried leaving these patients alone generally, with operative interference only in some cases in which symptoms seemed to require surgery. Various measures have also been employed—repeated tapping, insertion of tubes and exploration. None of these seems proper as a routine procedure. Each patient must be individualized in terms of the injury that he receives and the type of injury being treated.

One cannot be sure merely by placing a tube in the chest that there is not a great deal of clotted blood, with continued bleeding, or empyema, or that decapsulation will not be required subsequently.

Each patient has to be individualized. If there is any possible reason for major injury to the lung, heart or intrathoracic structures, exploration must be performed. On the other hand, there are many cases in which I am sure this procedure would be quite meddlesome.

This is not the same problem in relation to exploration that we find in the neck or situations overlying vessels. It is not the same problem we encounter in relation to abdominal stab wound injuries. Penetrating wounds of the abdomen must be explored because the possibility of a major fatal injury being present cannot be ruled out.

Injuries to the chest can usually be evaluated to determine the type of damage, and the patient can be treated accordingly. Therefore, we individualized these patients very thoroughly. However, penetrating abdominal injuries and those over or near the sites of large blood vessels cannot be individualized in the same manner.

LESTER BREIDENBACH (New York, N. Y.): Because of our inability in many instances to make an accurate diagnosis before exploration is performed, most of these papers have made a plea for early exploration in order that the diagnosis could be made and necessary definitive surgery then performed.

The authors of the paper on wounds of the neck have brought to our attention this somewhat neglected subject, from the standpoint that many of these neck wounds have not been explored in the past and the mortality rate has been considerably higher than it should be.

A lot of "real estate" is crowded into a small area in the neck and a penetrating wound, whether it be a knife wound or bullet wound, can do untold damage. I think it has been brought out very efficaciously that the amount of damage and its location is almost undiagnosable by any clinical or laboratory means we have.

I believe all such patients should be explored, as the authors suggested. When this has been performed mortality rates have shown a marked decrease.

I would also like to add that an adequate incision should be made so that exploration can be very thorough. I believe that the collar incision we use for thyroid, raising the flaps good and high and cutting the sternocleidomastoid muscles on each side when necessary, gives a very wide exposure of the neck and a large exposure of every structure. Necessary definitive surgery should be performed in these instances because the great majority of these wounds inflict vascular injuries which should be repaired at the time of exploration.

JAMES C. DRYE (Louisville, Ky.): The authors of the paper on wounds of the neck certainly brought out a very important subject, and it was very well treated.

Regarding chest injuries, I do not believe the experience of these authors has paralleled ours. We treat a great number of patients with chest wounds in our large city charity hospital, and we certainly do not operate routinely on them. I agree with Dr. Johnston that they should be individualized.

Generally, you can determine fairly well what the extent of the injury is likely to be. If it is a bullet wound, there is a wound of entrance, and either a bullet seen on x-ray or a wound of exit. By projecting lines one can determine where the bullet has been.

Knife wounds are somewhat more difficult. The path of the weapon cannot be determined as accurately as with a bullet wound since usually there is only the wound of entrance. As a rule, we do not explore wounds of the chest unless we have evidence of continued bleeding or suspect that the missile may have penetrated the diaphragm or traversed the mediastinum.

I think if we operated upon all of our patients we would have a great number in the hospital for quite a while, because we treat two or three such injuries a week.

The mortality rate was quoted as 10 per cent. I should like to ask the authors how many patients died from conservative treatment and what was the cause of death in each case.

GEORGE B. SANDERS (Louisville, Ky.): I would like to emphasize the great value of tracheotomy in the management of these patients who have excessive bronchopleural secretions which they have great difficulty eliminating.

JOHN PAUL NORTH (McKinney, Tex.): Drs.

Fogelman and Stewart have given us a magnificent discussion of this subject of neck wounds, and we must accept their thesis that penetrating wounds of the neck must be explored just as wounds of the peritoneum are explored.

They have mentioned tracheostomy, which came up several times in the discussion of management of various types of injuries and of burns.

I would like to comment on this. In principle, the procedure of tracheostomy is so absurdly simple that we assume anyone can perform it. In practice, and under the circumstances in which it must often be performed, it is an operation which requires much surgical dexterity.

We spend a great deal of time instructing residents in how to perform gastrectomy, but we assume that by reading a few paragraphs in a book they will know how to perform tracheostomy. Then, when they first undertake this procedure, it is under circumstances of haste, with poor illumination, difficulty in positioning the patient, unskilled assistance and often an inadequate armamentarium of instruments.

I believe we should make sure that all residents and interns who pass through our hospitals have a thorough opportunity to know how to perform tracheostomy. In view of the fact that one can never predict when this occasion may arise, we ought to go back to the medical schools and insure that medical students know more about it than I think they commonly do.

Most of us will agree that in general we should not teach operative technic to medical students, and yet, the operation of tracheostomy is one in which the details should be thoroughly understood before the need arises.

There is one other point in this respect. I think there is need for a great deal of improvement in the instruction of nurses and other personnel in the wards in the management of the post-tracheostomy patient.

I find profound ignorance in such simple matters as the maintenance of a free airway by aspiration of secretions, the need under certain circumstances for humidification of the air, and the very fundamental factor that a tracheostomy patient is not able to call for help if he needs it in a hurry. He should have some very practical way of summoning assistance if he is not constantly attended.

Richard L. Rapport (Flint, Mich.): Since previous discussors have been in disagreement with Drs. Sheesley and Roettig I should like to say a word in their support. I hesitate because Dr. Johnston was my Professor of Surgery, and I am very glad at this point he can do nothing to me academically.

We in Flint find difficulty interpreting the extent of trauma to the chest from either physical or radiographic findings. We have been universally surprised at the relative severity of the chest injury in patients subjected to thoracotomy, as compared with x-ray and clinical findings.

In all sincerity, we have yet to regret opening a chest for trauma, but on many occasions have censored ourselves for non-operative management. In open chest injuries hospital stay has been shortened, return to work hastened and complications reduced by thoracotomy. Indeed, we wonder whether or not more *closed* chest injuries should be treated by operative measures.

Howard Errol Snyder (Winfield, Kan.): I am usually in the position of Dr. Roettig—that of being criticized for opening too many chests or being too radical in treating them. Now I am in the unusual position of joining Drs. Drye and Johnston who have criticized the statement that stab wounds and penetrating wounds of the chest should be subjected to thoracotomy.

To elaborate further, we finally got those who were criticizing the work done in the forward hospitals to agree with us that there were certain indications for thoracotomy. They include diaphragmatic injury, right or left, and suspected diaphragmatic injury. When there is a possibility of injury to the left diaphragm, there is the possibility of perforation of a hollow viscus, just as there is when the peritoneum is perforated. Therefore, operation should be performed in some questionable cases to insure safety.

I believe that the same applies to the right diaphragm because the complications which may ensue from biliary fistulas and the like warrant that exploration. Dr. Drye mentioned this indication. He also mentioned continuing massive hemorrhage. By that we do not mean continuing hemorrhage that requires aspiration twice the first day and only once daily thereafter. Those chests, if managed conservatively, certainly must have repeated thorough evacuation of both blood and air from the pleura.

Beside the esophageal or mediastinal injury Dr. Drye mentioned I would add the indications of large tracheal or bronchial fistulas, lungs which do not expand with an intercostal tube, pleural foreign bodies regardless of size, and large foreign bodies retained within the lung.

Louis C. Roettig (closing): To close the discussion of our paper, I thought that I perhaps could better explain our position in regard to the selection of patients with penetrating wounds of the chest for operation than could Dr. Sheesley, since it is primarily my responsibility.

Dr. Breidenbach expressed our opinion accurately when he stated that in all probability surgeons cannot make as accurate a diagnosis in many of these cases as some of us have believed possible.

In reply to Dr. Johnston, let me say that we do not mean to imply by this paper that we explore

every penetrating wound of the chest. We do believe that the majority of these patients, however, merit exploration, and by means of this presentation we wish to urge a more aggressive attitude toward the management of these wounds. We believe that it is impossible to make as accurate a diagnosis in these cases as was formerly thought.

This inexactitude in our diagnostic ability can be illustrated by two cases, one of which served as an introduction to our paper. The second case will answer Dr. Drye's query about the two patients who died following conservative management. Both of these patients showed transection or laceration of large pulmonary vessels which was not accurately diagnosed at the time of admission. I will cite just one of these cases.

A man came to the Emergency Room with a penetrating knife stab wound in the chest. I cannot recall the exact interspace level. After he had been stabbed he shot his assailant in the abdomen. Both men were brought simultaneously to the Emergency Room of White Cross Hospital. The man who had sustained the stab wound of the chest was ambulatory; the man who had been shot in the abdomen was carried in and treated by the members of the house staff. A chest x-ray of the man with the stab wound showed only a small amount of pneumothorax and practically no fluid in the chest. He was admitted to the hospital and placed in bed. Several hours later he was found in profound shock. It was impossible to reverse this condition, or for the staff to get him to the operating room before he died. Autopsy revealed almost complete transection of the inferior pulmonary vein on the left side.

It is cases such as these which have brought us to decide that more of these patients with penetrating wounds of the chest should be explored. We have come to question quite honestly the accuracy of our own or anyone else's best judgment regarding these wounds. Certainly, the treatment of such a patient should be individualized, and the surgeon should make every attempt to evaluate the patient as carefully as possible before deciding upon the mode of therapy. However, we believe that a more aggressive attitude in therapy is needed.

ROBERT STEWART (closing): We are very grateful to the gentlemen who discussed our paper. I thought their remarks were clear and pertinent as well as additive to our premise.

Dr. Breidenbach mentioned a matter of incision for exploration of the neck. In performing explorations in the present series, several incisions have been utilized. The oblique incision over the anterior border of the sternocleidomastoid with retraction of a muscle laterally has been completely satisfactory for unilateral exploration for approach to the esophagus and pharynx, and drainage of the cervical mediastinum.

In transverse wounds the collar incision gives excellent exposure. In many knife wounds, exploration has been performed through the original wound with extensions. However, exposure is of paramount importance in these wounds, and in some cases these extensions must be rather generous.

Our approach to the base of the neck and to the superior mediastinum has been a superior one with partial resection of the clavicle.

In addition, there may be occasions in which the sternum must be split to gain access to mediastinal structures, or thoracotomy may be indicated in some cases in which any immediate proximal control of the major vessel is necessary.

Heart Wounds—A Long-term Follow-up of Twenty Cases

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THE drama of the acutely wounded heart has understandably caused surgeons to focus their attention on immediate results, i.e., life or death. Large series of cases reported by Griswold and Maguire,[1,2] Griswold and Drye,[3] and Elkin and Campbell[4] have been devoted to this aspect of the problem. Griswold has been the principal advocate of immediate pericardiotomy, and Elkin the advocate of treatment by pericardiocentesis, reserving pericardiotomy for selected cases. One cannot choose between these two types of treatment on the basis of mortality rates because of the small differences and because of the presence of important variables other than those of the form of treatment. There is a surprisingly high rate of salvage with either type of treatment. In our series 75 per cent of all patients operated upon lived, and even more important, the gross uncorrected survival rate of all patients admitted was 62.1 per cent. This latter figure includes all patients who showed any manifestation of life when first seen in the emergency room.

In this paper we focus our attention on the long-term results obtained in our series. All of these patients were treated with pericardiotomy.

From 1934 through 1951 there were sixty-five survivors. We have been able to study only twenty of these. (Table 1.) This meager percentage of followed cases is not surprising to anyone who has worked with the usual type of individual who is stabbed or shot. Such persons are not noted for their sobriety, steady working habits, or friendliness with the police or others who search for them. The twenty patients were selected in the sense that they were either those we could find and persuade to come to the hospital for study or those who came back because of illness. One of the authors (W. S. C.) interviewed and examined all of the twenty patients. Evidences of heart disease, such as subjective symptoms, limitation of activity, and physical signs, were carefully sought. Chest films and electrocardiograms were taken routinely.

TABLE 1

Total number of survivors	65
Number followed up	20
Number with heart disease	6
Number without heart disease	14
Number working	12
Number not working	8

The length of time of follow-ups varied from three to twenty years. Ten of these patients had been injured ten or more years before the time of the study.

Twelve of these patients are now working; seven of these twelve have no symptoms and no evidences of heart disease.

The remaining five patients who are now working do have symptoms: Two have definite heart disease. One of these is now seventy-five years old, works as a porter and has definite evidence of arteriosclerotic heart disease. He sustained his injury when sixty-six years of age. The other, a woman, was thirty-four years of age when she was injured; at the time of her last examination she was thirty-nine years old. She has definite hypertension with left ventricular enlargement by x-ray, and left ventricular ischemia by electrocardiographic studies. She has mild anginal attacks; however, she continues to work in a fish market. Her injury was a stab wound high in the right ventricle.

The remaining three patients who work have vague chest pains and questionable dyspnea, but none shows any evidence of heart disease.

Of the eight patients who are not working, four have definite heart disease. One man, who had a laceration of the right ventricle at the age of thirty-eight, was last seen in 1953 when he was fifty-two. At this time he was found to

have hypertensive cardiovascular disease with severe decompensation. His blood pressure was 180/106 and he had x-ray evidence of left ventricular enlargement and electrocardiographic evidence of an old posterior wall infarct. Another of these non-workers with heart disease was sixty years old when he sustained a penetrating wound of the right ventricle. Previously he had done heavy work, but since his injury he has not tried to work. At present his only complaint is that of a slight cough. Examination at our hospital before his injury had revealed an enlarged left ventricle by electrocardiography and x-ray studies, a blood pressure of 172/78 and a capillary pulse. The next patient, stabbed in the left auricle in 1951 at the age of forty-nine, had a blood pressure of 168/120 in 1953 but shows no other evidence of heart disease. However, he does complain of some dyspnea and has not worked since he was wounded. The last of these four patients was shot in the spine, with resulting paraplegia, seven years after his heart wound. He also had definite luetic heart disease.

In four of the eight non-workers we can find no evidences of heart disease. Two of these are young men; one just does not feel like working, and the other tried to work as a garbage-tipper but found it too strenuous. Another patient worked for seventeen years but had a cerebral hemorrhage with hemiplegia and has not worked since. The fourth man, aged fifty-three, did work off and on for a time but has not worked for the past five years. He believes that he is too old.

COMMENTS

Six of the twenty patients that we were able to trace from a total series of sixty-five survivors of heart wounds were found to have definite evidence of heart disease. We believe that the unusually high number of cardiac patients in this series is due to a process of selection. As previously mentioned, it is usually rather hard to keep up with the type of person who suffers a heart wound. However, when one of these patients becomes ill, he will come into the hospital; and in this type of case the patient will very likely come back to us as ours is the only charity hospital in the city.

In studying these patients we were particularly on the lookout for the type of heart dysfunction that would most likely occur after a heart injury, that is, such things as disorders in rhythm, local aneurysmal dilatations, constrictive pericarditis and possibly a picture resembling that of cardiac infarction. Only one patient was thought to have an infarct. He had been stabbed in the anterior surface of the right ventricle and the infarct was presumed to be on the posterior surface according to electrocardiographic studies. He was located twelve years after his injury, and at this time he was fifty-two years old. We do not believe this infarct was related to his heart wound.

There were no findings in any patient that could be attributed to the wounds. Certainly no evidence of constrictive pericarditis was demonstrated in any case.

In the recent literature we have found seven cases of constrictive pericarditis following hemopericardium due to blunt and penetrating wounds, which were treated by aspiration.[5–11] In 1909 Rudolph Matas[12] commented on this possibility and reported two such cases. Even if the blood remains liquid—and we have removed large clots on many occasions—it would seem impossible to remove all of it; also, as in the pleural cavity, the remaining blood may not be completely absorbed and this could lead to a constrictive fibrous capsule. This probably explains the occurrence of constrictive pericarditis which has been reported following aspiration of blood in the pericardium.

CONCLUSIONS

1. Twenty patients with stab wounds of the heart have been followed up for periods ranging from three to twenty years.

2. In six of these patients heart disease developed which could not be attributed to the stab wounds. The various conditions represent usual types of organic heart disease, namely, that due to hypertension, arteriosclerosis and, in one case, syphilis.

3. There have been no cases of constrictive pericarditis or false aneurysms.

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Practical Aspects in the Management of Stab Wounds of the Heart

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The management of stab wounds of the heart is a challenge to our present knowledge of cardiorespiratory physiology. In the past the mode of treatment was based upon a small amount of individual experience and an extensive review of the literature. Our textbooks had perpetrated the prejudice that the common cause of death in stab wounds of the heart was cardiac tamponade. Furthermore, the use of intravenous fluids or blood was delayed on the precedent that such therapy would further increase the undesirable elevation in venous pressure. In the past attention was directed mainly to the right heart and the associated circulatory stagnation, even though shock was recognized in these patients. At present it is difficult to understand how shock was so studiously neglected. Today emergency therapy is designed to support the left heart and the systemic circulation.

The growth of modern cardiac surgery has given the surgeon a more courageous attitude toward the management of stab wounds of the heart. Pericardicentesis was the immediate and usual solution to the problem in many cases of tamponade, but recently surgical exploration has been performed with increasing frequency. Accurate observations made at the operating table have revealed many of the fallacies of our former therapeutic management. As a result of these surgical adventures it is evident that cardiac tamponade *per se* as the cause of death is difficult to confirm. However, in some cases tamponade as a contributory cause of death deserves consideration. At the present time the main lethal etiologic factors are thought to be due to (1) alterations in the coronary circulation, (2) hemorrhage and (3) changes in pulmonary physiology.

PATHOLOGIC PHYSIOLOGY

An intact coronary circulation is essential to the survival of an injured heart. Sudden death from myocardial anoxia is the *usual* result subsequent to traumatic interruption of the coronary artery or one of its significant branches. In general, it may be assumed that the patient who reaches the hospital has sustained no *significant* anatomic damage to the coronary system. The patient with injury to the heart is almost always found in a state of shock or circulatory collapse, or both. Since the coronary arteries are a part of the systemic circulation, the status of the coronary flow is but a reflection of the systemic circulation. Diminished circulation in systemic blood vessels implies sluggish circulation in the coronary system which implies that myocardial anoxia is imminent. In the presence of shock myocardial anoxia may prove to be more serious than the injury itself.

We know from our experience in modern cardiac surgery that the heart tolerates the most unusual surgical trauma in the presence of normal coronary circulation. This suggests that in cardiac injuries not only must the anatomic interruption of the coronary circulation always be considered, but also the "physiologic interruption" of coronary flow which is secondary to collapse of the systemic circulation. In view of the fact that the coronary arteries fill during diastole it is evident that coronary flow is dependent upon the diastolic pressure in the ascending aorta. The factors mainly responsible for diastolic pressure are (1) total blood volume, (2) peripheral resistance and (3) cardiac output. All three are closely related, and in the patient with a cardiac injury all three are functioning to the patient's disadvantage. Therefore, it is desirable to obtain immediate improvement of the coronary circulation in order to insure additional time for cardiac evaluation as an aid to the surgical decision.

When the injured patient is encountered in the emergency ward, the examining physician

does not know the cause of circulatory collapse. It may be an "anatomic interruption" of the coronary system or "physiologic interruption" of the coronary flow, secondary to the shock and generalized circulatory collapse. The differentiation between the two will be made by the response to therapy designed to increase the blood pressure in the ascending aorta and consequently, an increase in the coronary flow. When significant coronary vessels have actually been severed ("anatomic interruption") therapy directed to improve the nutrition to the anoxic area of the heart will be ineffective. In distinct contrast is the response of the heart with an intact coronary system but in which the myocardium is suffering from the anoxia of reduced coronary flow ("physiologic interruption"). This heart will improve as a result of therapy directed at shock if the process is still reversible. The exception to this premise occurs when, in addition to the injured heart, there is also damage to the lung. Under these circumstances the blood composition (CO_2 and O_2) is qualitatively deranged. It is evident that the injured patient with collapse of one lung or partial collapse of both lungs supplies the left heart and coronary vessels with blood high in CO_2 and low in O_2. It follows that myocardial anoxia persists even though the coronary circulation is adequate. Therefore, therapy must be directed to the correction of the cardiorespiratory physiology.

EXPERIMENTAL STUDIES*

In the experimental laboratory healthy dogs which had been anesthetized were utilized in experiments simulating stab wounds of the heart. Scissors and sharp scalpels were used as the penetrating instruments and attempts were made to create artificial tamponade by closing the opening in the pericardium. The left auricle, the left ventricle and the intrapericardial portion of the aorta and pulmonary artery were the sites selected for injury. To be practical only small openings, the size of the penetrating instrument, were made. It was reasoned that large wounds in the ventricle or aorta would not be instructive because such rents and the associated massive hemorrhage into the pleural cavity would result in immediate death by exsanguination. Patients with similar injuries in civilian life rarely reach medical attention.

Experiments. Type I: The left ventricle was stabbed as many as eight times with a very narrow sharp scalpel (0.5 mm.), but the coronary arteries and significant branches were always avoided. The spurting blood soon clotted and the openings were closed within ten minutes. Tamponade did not develop.

Type II: The tip of the left auricle was excised with a pair of scissors and the pericardium was closed. Tamponade did not develop although the pericardium filled with blood.

Type III: A small rent, 0.5 cm. in length, was made in the anterior wall of the pulmonary artery with a sharp pair of scissors. Tamponade did not develop. A clot formed in the defect.

Type IV: A sharp scalpel (0.5 cm.) was plunged into the intrapericardial part of the aorta. Bleeding was profuse; the pericardium became tense with blood. The heart rate increased slightly. Ten minutes later the pericardium was opened, the clot evacuated and the opening was found to be closed by clot formation.

Type V *and* VI *combined:* The damage was done by a single plunge of a sharp pair of scissors, creating a fistula between the aorta and the pulmonary arteries with severe bleeding into the pericardium. Death did not occur in four hours of observation, although the pericardium was tense with blood.

Type VII: The anterior descending branch of the left coronary was divided with scissors at its origin. Death occurred within fifteen minutes. Ligation of this artery in dogs causes death in 70 per cent of the cases, but when hemorrhage superimposes myocardial asphyxia death is the usual result. Death always occurs if the left coronary artery or its circumflex branch is divided.

PRACTICAL MANAGEMENT

Patients who sustain penetrating injuries to the heart may be divided arbitrarily into two great groups: (1) those who die almost immediately and (2) those who arrive at the hospital in various degrees of shock. In the first group the insult is of maximum intensity. These patients usually die from exsanguination due to *large* rents in the ventricle or major vessels at the base of the heart.

Patients who survive to reach medical attention are usually in shock secondary to altera-

* Experimental studies carried out in the laboratories of Hoffmann-La Roche, Inc., Nutley, New Jersey.

tions in cardiorespiratory physiology, hemorrhage and concommitant trauma. As these patients enter the emergency ward a cursory examination discloses that they fall into two groups. In group 1 the injury is so mild and the patient's condition so good that close observation in the emergency ward for twelve hours is all that will be necessary. In group 2 the injury and the general condition of the patient are moderate to severe and since the former may progress to the latter, the vigilance and therapy must be essentially the same. Therefore, patients in group 2 deserve an orderly and precise pattern of management.

A *history* from patient, friend or witness may prove invaluable. The time of the injury is important in estimating the duration and reversibility of the shock. The nature of the instrument and axis of force are helpful in estimating the extent of damage and potential infection of the underlying structures. Tamponade is less likely to develop if the traumatizing instrument makes a large rent in the pericardium, because blood may escape (1) externally, (2) into the mediastinum or (3) into the adjacent pleural cavity. It does not follow, however, that tamponade is assured in the patient with a small rent in the pericardium, because hemorrhage from the small opening in the vessel, ventricle or auricle is easily occluded by clot formation. The patient's general health (e.g., diabetes, tuberculosis) and immediate condition (alcoholism) prior to injury may help in the clinical evaluation.

The *physical examination* stresses the general appearance, vital signs, status of the cardiorespiratory system and the location and character of the major wound or wounds. The general appearance of most of these patients is consistent with the picture of shock. They are pale, clammy, cyanotic and, if conscious, anxious and restless. The neck veins may or may not be distended, depending upon the degree of circulatory collapse. In some cases the extremely reduced volume of blood returning to the heart may be insufficient to distend the neck veins even in the presence of cardiac tamponade. In reviewing the vital signs, the pulse is usually rapid and thready. A narrowed pulse pressure and a paradoxic pulse strongly suggest the presence of tamponade. The blood pressure may be critically low and this examination should be repeated frequently in order to establish a base line for the therapy which follows. The status of the cardiorespiratory system is easily determined by a thorough physical examination of the chest with the patient in a sitting position. Tracheal or bronchial rhonchi may be caused by blood in the airways. The rate and depth of respiration may be due to the accumulation of CO_2, tracheobronchial secretions, anoxia or alcoholism. Distant heart sounds and a quiet heart suggest tamponade. The location of the wound is significant because of possible injury to other vital structures and organs, such as the esophagus, liver, spleen, and mammary and intercostal arteries. Examination of the character of the wound is designed to disclose external bleeding and the presence of a sucking wound. A general survey of the entire body will exclude other causes for the clinical status.

The *practical management* actually begins as soon as a diagnosis of shock has been made from the physical examination and obtainable history outlined previously. It is the duty of the first examiner to initiate shock therapy by improving, if possible, the cardiac output, correcting the blood volume and perhaps increasing the peripheral resistance. An infusion or a blood volume expander should be started immediately. Blood for typing and cross matching may be obtained through the same needle. A base line of the vital signs should be established from the moment of arrival, and recorded at frequent intervals. An adequate airway, oxygen therapy and occlusion of the sucking wound on expiration will improve alterations in the respiratory mechanism. Medication should be used for pain and the misinterpreted restlessness of an uncooperative or struggling patient, since straining reproduces the Valsalva principle which interferes with the return of blood to the heart and indirectly lowers the cardiac output. The administration of oxygen is the best adjunct when the restlessness is on the basis of air hunger.

Once the infusion has been started and a portable oxygen unit made available, the patient may be moved to the operating suite or the adjacent recovery room where all facilities are available for further evaluation and treatment. At this moment the radiology department, previously notified, will take a portable roentgenogram of the chest in the upright position. Equally important is the early notification of the operating room supervisor, anesthetist and surgical team to the possibility of a

major operation. In the operating room there should be equipment available for aspiration of the chest or pericardium, a set-up for closed or open thoracotomy, the essential instruments for tracheostomy and the electrical apparatus for cardiac resuscitation. The precision of the treatment instituted in these early moments is critical and we agree with those who believe that a speculative attitude at this point is the sire of irreversible shock. However, certain general information may constructively contribute to the surgical decision. First, and foremost, is the possibility that the coronary artery or one of its significant branches has been interrupted. Supportive therapy will *not* improve the nutrition to an ischemic area of the heart if the penetrating instrument has severed an important branch of the coronary system. Furthermore, nutrition is not improved by a surgical suture to control bleeding from such a vessel. On the other hand, it is encouraging evidence to see a patient respond to intravenous therapy, for this implies that the main coronary circulation is intact. Furthermore, it suggests that the state of shock may be dependent upon a marked reduction in blood volume (hemorrhage) or significant interference with pulmonary physiology. Hemorrhage and pulmonary insufficiency are the constituents responsible for "physiologic interruption" of the coronary circulation. As a result of hemorrhage, the volume of circulating blood is inadequate for myocardial sustenance which together with partial collapse of a lung and the associated disturbance in the exchange of oxygen, provides the intrinsic circulation of the heart with blood qualitatively inferior (CO_2 and O_2). Therefore, quantitatively and qualitatively the coronary circulation may be inadequate in the presence of a severe hemopneumothorax. An immediate portable chest roentgenogram with the patient in the sitting position will confirm the physical findings of a hemopneumothorax, and provide a control for additional roentgenograms if continued bleeding is suspected.

To be practical, the chest roentgenogram is of tremendous importance. If severe shock is present and the chest roentgenogram does not disclose a significant hemothorax, then the patient's condition is probably not due to hemorrhage but to tamponade, or actual injury to the coronary circulation. If distended neck veins or other evidences of tamponade are present, immediate pericardicentesis may be life-saving. These patients must be followed very closely if they improve following aspiration, since a second pericardial decompression is frequently necessary. Should the patient relapse again after the second pericardial aspiration, continued bleeding is suspected and a third aspiration is performed preparatory to open thoracotomy. If the chest roentgenogram reveals a *large* collection of blood in the pleural cavity and the lung is collapsed in proportion, then the injury to the heart or significant vessels is a major one and surgical exploration is imminent. Intravenous fluids or blood provide only temporary security in such cases. Recurrent hemorrhage is embarrassing to both patient and surgeon. Delay in performing open thoracotomy is justified in selected cases when age, other insults and associated disease so dictate. As a general rule, however, with sufficient blood, good anesthesia and a competent surgeon the risk of surgical exploration is less than the risk of procrastination.

SUMMARY

1. Patients sustaining a stab wound of the heart, in which significant branches of the coronary artery are divided, rarely live long enough to receive medical attention. Should the patient survive such an injury, a hasty hemostatic suture placed by an eager surgeon will not improve the nutrition to the anoxic myocardium distal to its ligation. The results from occlusive disease of the coronaries are quite analogous.

2. Furthermore, patients with *large* rents in the cardiac chambers, or major vessels (within the pericardium), rarely survive to reach the hospital because these patients also have large rents in the pericardium. Hemorrhage, not tamponade, is the cause of death in such cases.

3. An emergency portable chest roentgenogram records and confirms the physical findings of a severe hemothorax as the cause for shock. A severe hemothorax implies *significant* damage to a cardiac chamber, structures in the hilum of the lung or a significant blood vessel (aorta, mammary or intercostal). Blood replacement provides security for surgical exploration, and surgical exploration provides security for the patient. In the presence of a severe hemothorax, closed thoracotomy may be done as a preparatory procedure to relieve pulmonary collapse which will aid the anesthetist during

induction prior to thoracotomy. Blood "outside" the lung has no physiologic function.

4. The work of Cooley and DeBakey demonstrates the practical importance of treating these patients by the immediate use of intravenous fluids or blood. In so doing, cardiac output is improved and the diminished blood volume is corrected.

5. The role of tamponade as the cause of death has been overemphasized. As a contributing factor it must not be overlooked. Its presence does not contraindicate the immediate use of intravenous fluids, plasma expanders or blood transfusions. In addition to shock therapy, decompression of the pericardium in the presence of tamponade provides *time* for additional studies and more accurate evaluation of the patient's condition.

6. Routine thoracotomy for a hemopericardium is not necessary. Antecedent trauma or a hemopericardium as etiologic factors in constrictive pericarditis are rarely reported. Modern cardiac surgery and experimental work on the surgical management of coronary insufficiency tends to minimize the hazards of a hemopericardium.

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Rupture of the Pericardium with Complete Extrusion of the Heart Resulting from a Steering Wheel Injury

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TRAUMA to the thorax may result in a variety of injuries involving not only the bony structures but also the intrathoracic organs, as well as the diaphragm and great vessels. Many of the chest injuries encountered in civilian practice are the result of car accidents. Drivers of motor vehicles usually sustain thoracic injury when their bodies are thrust forward against the steering wheel. Such injuries are known as steering wheel injuries to the chest.

Rupture of the pericardium from a steering wheel injury is apparently a rare occurrence. In two cases[4,5] the pericardial ruptures were found and recognized only at necropsy. In both instances the heart was incarcerated within the pericardial rent, resulting in cardiac standstill and death. A similar condition has been reproduced in the experimental animal (dog) in our laboratory.

The present report concerns a patient who survived complete extrusion of the heart following rupture of the pericardium.

CASE REPORT*

On November 7, 1954, a forty-four year old white man, who suffered a steering wheel injury to the chest, was admitted to the hospital. He had fallen asleep at the wheel and his car collided head-on with a tree.

Examination on admission revealed the following data. The principal complaint was chest pain on deep respiration. There were multiple lacerations on the face and right knee. Blood pressure was 170/100. Coarse rhonchi were heard bilaterally with dullness on percussion at both bases. Depression of the lower third of the sternum was noted. The thoracic cage was stable. Heart sounds were distant. There was sinus rhythm and the apical rate was 105 per minute. There was no evidence of cardiac enlargement or displacement.

X-ray examination of the chest revealed depression of the lower third of the sternum and fractures of the left fifth and sixth and right fourth, fifth and sixth ribs. Lobular atelectasis was noted in the lower half of the right lung and a collection of pleural fluid was present at the base of the left lung. There was no mediastinal shift.

Admission electrocardiogram demonstrated clockwise rotation of the heart, sinus rhythm, a rate of 107 per minute, and diphasic T wave in aVF suggestive of posterior myocardial damage.

Laboratory findings were within normal limits.

The patient was treated conservatively with bedrest, chest taping, a Levin tube connected to suction to alleviate gastric distention, nothing by mouth (twenty-four hours), intravenous fluids and narcotics for pain. Weekly roentgenograms and electrocardiograms were taken. The Levin tube was removed after seventy-two hours and the patient started on oral fluids.

The patient continued to improve with clearing of rhonchi in lung fields. He complained of chest pain only on deep inspiration. The heart sounds remained distant with the rate varying between 120 and 98 per minute. The blood pressure fluctuated between 170 and 98 systolic, and between 100 and 70 diastolic.

On November 12, 1954, the patient complained of severe pain in the left great toe which was red, swollen and painful on movement. X-ray of the toe was negative. However there was elevation of blood uric acid and a diagnosis of gouty arthritis of the left great toe was made. The patient was placed on purine-free diet and colchicine. He was unable to tolerate the latter and medication was changed to sodium salicylate to which he responded.

The patient's hospital course remained uneventful except for the electrocardiographic changes.

Chronologically they were as follows: November 8, 1954: Since November 7th T waves V_2 inverted, Q waves in aVF suggestive of posterior infarction; marked clockwise rotation of heart still evident; rate 115. November 13th: No significant change

* Courtesy of Dr. James M. Kennary, Detroit, Michigan.

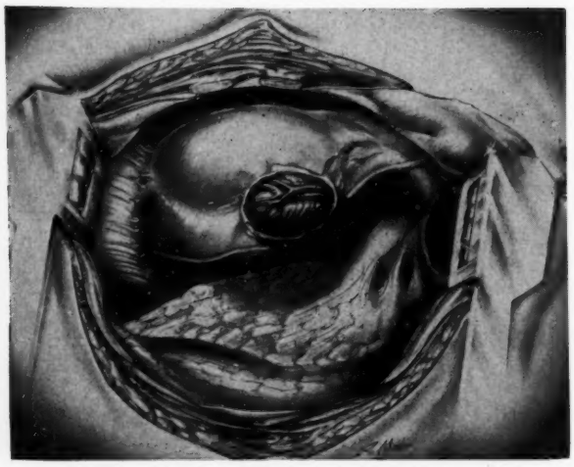

FIG. 1. Drawing of a small pericardial rent resulting in cardiac incarceration and death. Observation at necropsy.

since November 8th; rate 110. November 26th: No significant change in standard and unipolar limb leads since November 13th; some improvement since November 8th in curve with T waves in $V_{2,3}$ now upright; marked clockwise rotation still present; rate 110.

Fluid in the left base persisted and there was roentgen evidence of organization of the fluid after three hospital weeks. Exploratory thoracotomy with the intention of performing decortication of the left lower lobe was decided upon. On December 3, 1954, the left hemithorax was explored. The heart, absent of its pericardial sac, was found beating freely in the left hemithorax. There was approximately 600 cc. of serosanguinous fluid in the left pleural cavity. The parietal pleura was greatly thickened with fibrin deposits as was the dome of the diaphragm. A pericardial tear, extending from its diaphragmatic attachment to the left atrial appendage, was found. The rent was above the phrenic nerve. The parietal pleura in the region of the torn pericardium was perfectly smooth. There was no evidence of a penetrating fracture of a rib edge. Careful examination of the epicardial and subepicardial surfaces of the heart revealed normal color and uniform consistency. There was no gross evidence of bruises, hemorrhage or injury to the heart. After inspection the heart was placed back into the pericardial sac and the rent repaired with No. 000 interrupted silk sutures. (Figs. 1 to 3.)

The patient's postoperative course remained uneventful. The most significant change was in the postoperative electrocardiogram taken on December 4, 1954 which showed the following: Definite change in position of the heart; No longer clockwise rotation; Left axis deviation and intermediate position evident with transitional zone now in V_4; diaphasic T waves V_{5-8}; aVL with straightening and slight depression of S–T segment in these leads indicate left ventricular subendocardial damage; T wave VE now upright; Q waves not present in aVL or left ventricular precordial leads, but still evident in aVF; rate 115.

Electrocardiograms taken on December 15th and 30th showed no essential change except the fall in rate to 80 to 85.

The patient was discharged from the hospital on December 17, 1954. His blood pressure stabilized around 150/100 with an apical rate of 80. Heart tones have continued to be of good quality with sinus rhythm.

On April 4, 1955, when a repeat tracing was taken, although slight sagging of the S-T segment in aVF was still present, definite electrocardiographic improvement was noted, as evidenced by return of a more normal contour of the S-T segment in V_{4-6} as well as return of the T waves to an upright position in these leads. (Fig. 4.)

COMMENTS

Two cases of ruptured pericardium resulting from steering wheel injuries have been obtained from the literature,[4,5] and one from the personal files of the author. The reported cases of Beck and Patton presented a similar clinical picture of acute compression of the heart (high venous pressure, low arterial pressure and a quiet heart). The case reported herein clinically resembles the cases of Beck and Patton except for the fluctuating arterial pressures and the marked clockwise rotation of the heart.

A patient who has sustained a steering wheel injury to the chest and presents a clinical picture of acute cardiac compression with a fluctuating or low arterial pressure and electrocardiographic findings with or without clockwise rotation of the heart, should be given the benefit of early exploratory thoracotomy. A larger number of pericardial ruptures from such injuries may then be recognized and successfully treated by surgery.

When complete extrusion of the heart is present from a large pericardial rent, the chances of survival may very well be good. Smaller rents of the pericardium are lethal lesions which can easily produce cardiac incarceration and death, as reported by Beck and Patton. The likely answer to this problem of steering wheel injuries or direct contusion to the chest with clinical evidence of acute cardiac compression is early emergency exploratory thoracotomy.

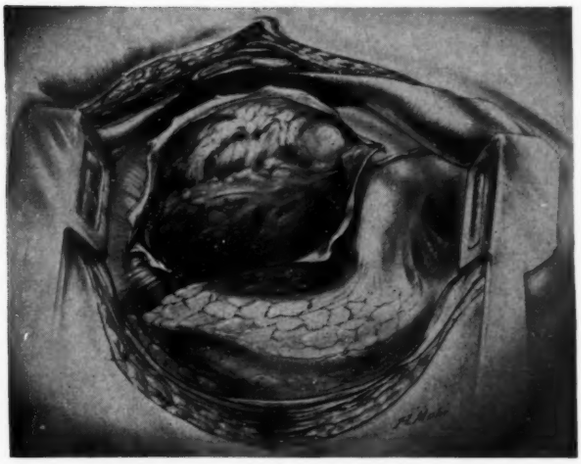

FIG. 2. Drawing of a large pericardial tear with complete extrusion of the heart. Observation at operation. Patient successfully treated.

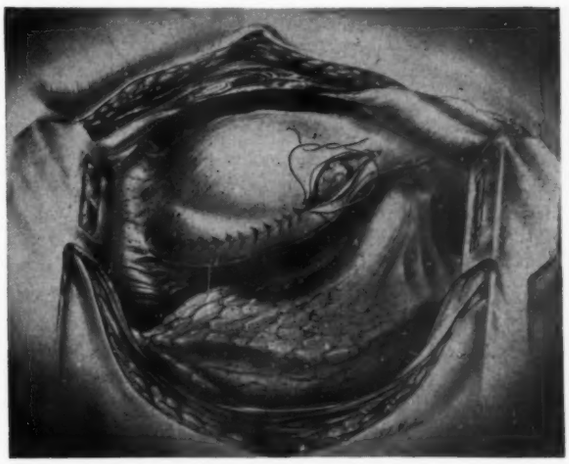

FIG. 3. Drawing of surgical repair of large pericardial tear with interrupted No. ooo silk sutures. Patient successfully treated.

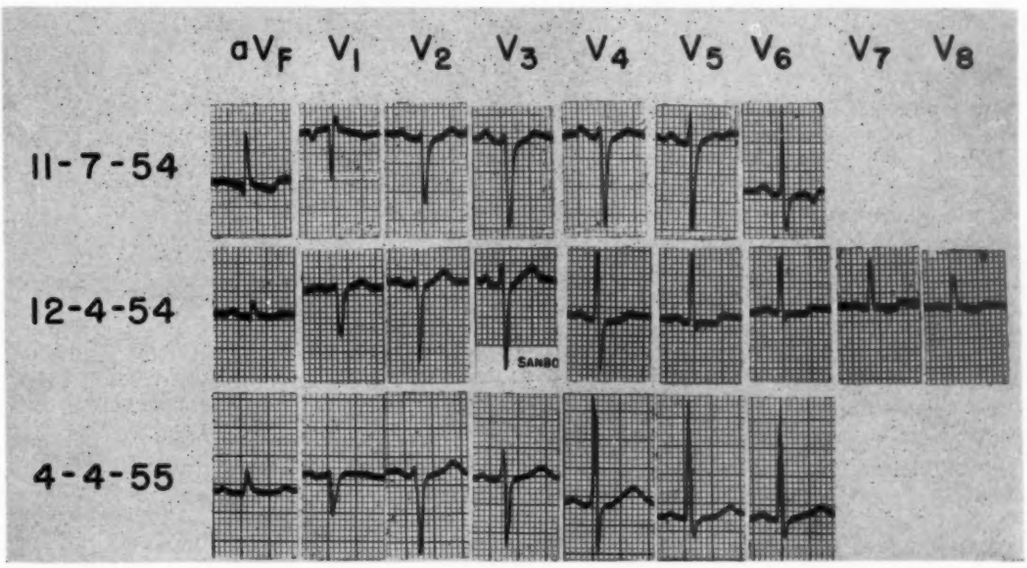

FIG. 4. Preoperative (11-7-54) and postoperative (12-4-54 and 4-4-55) electrocardiographic tracings. Note change in position of the heart in postoperative electrocardiograms. No longer clockwise rotation.

SUMMARY

A case of ruptured pericardium with complete extrusion of the heart resulting from a steering wheel injury, which was successfully treated surgically, is reported.

Acknowledgment: The author wishes to thank Dr. Fred Talmers of the Wayne University College of Medicine for the preparation, reading and interpretation of the electrocardiographic tracings.

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DISCUSSION OF PAPERS BY DRS. DRYE, COLE AND STAMER, CHAMBERLAIN, STEFKO AND CARBERRY, AND JOHNSON

EDWARD E. AVERY (Chicago, Ill.): Dr. Chamberlain's paper is a timely report which should encourage us to make a careful, but calm, evaluation of patients with penetrating wounds of the heart. The overemphasis of the dangers of pericardial tamponade has perpetrated many hasty thoracotomies. It should be obvious that the patients who reach the hospital alive probably do not have a wound through a major coronary artery and are capable of at least temporary vital function. Dr. Chamberlain has graphically illustrated that the heart's function can be supported without immediate surgical repair if adequate oxygenated blood is supplied to the myocardium. An upright chest x-ray should be taken on admission to evaluate possible pneumohemothorax and pericardial silhouette. Attention to a clear unobstructed airway will insure a supply of oxygen to the lungs, and blood transfusions will correct shock and provide adequate volume and pressures of oxygenated blood to the coronary vessels for vital function. At this point a sensible re-evaluation of the effects of the wound and tamponade can be made

The literature is replete with warnings of the adverse effects of acute pericardial tamponade. The conclusions are based on the often repeated dog experiment of Cohnheim, who in 1899 injected mineral oil under pressure into the fluid-tight pericardial sac to produce tamponade and cardiac failure. Each year a great number of dogs succumb to the effects of tamponade produced in this unphysiologic manner. By reproducing actual stab wounds of the heart the author has shown that the role of tamponade has been overemphasized. This work should stop further Cohnheim experiments and likewise make Dr. Chamberlain a candidate for "dogs' best friend of 1955."

Conservative management has been recommended in the past by Elkins, Griswold, Blalock and others. Blood replacement, pericardial aspiration and careful observation often is adequate treatment. This in no way should negate the role of exploratory thoracotomy in properly evaluated patients in whom this may be a life-saving operation.

I am sure that Dr. Chamberlain's work and recommendations will save some humans and a great many dogs.

WILLIAM H. CASSEBAUM (New York, N. Y.): I am rather reluctant to comment because my experience with heart wounds is very limited.

Dr. Chamberlain may have made a great contribution with his paper. However, the teaching of cardiac tamponade is so old that I am somewhat in the position of the farmer who saw one of the early locomotives pull away from the station and said he still didn't believe it.

There certainly has been a good deal of evidence in the literature that heart wounds do well on conservative treatment, either with aspiration or sometimes even without it.

Most of the foreign bodies removed from chambers of the heart during the war were done well behind the lines and many days after the wounding. Also, a great many of the operations for tamponade have been done rather late. So, while I remain confused, I certainly would be very hesitant now to advocate immediate opening of the chest because of a stab wound of the heart.

Dr. Chamberlain's presentation is certainly very dramatic. However, there are one or two points on which we might differ. One is that we can assume the coronary circulation is intact when the patient gets to the hospital. However, as we know, a great many patients with coronary thrombosis will live for four hours or longer.

Also, I think there must be some cases in which the wounding of the conduction mechanism must be of importance in the result.

R. ARNOLD GRISWOLD (Louisville, Ky.): As many of you know, I have been interested in this topic of heart wounds for a long while.

Regarding Dr. Drye's paper, I think the factor which amazed us about those patients who returned was the small incidence or the absence of cardiac disability which could be attributed to the wound.

There are obvious difficulties in following this type of patient. Because there is still a fairly high incidence of syphilitic cardiac disease, these patients do not return if a policeman or social worker is sent after them because a good many of them have a wife that they are not supporting and a social worker is just as bad in their eyes as a policeman. Therefore, the only patients who do return to the hospital are those who are sick for some reason or another.

I was very much interested in the paper by Drs. Chamberlain, Stefko and Carberry. They certainly reported an unusual accident and should be congratulated upon having finally recognized it.

Possibly we may have found a new diagnostic sign in the rotation as shown by the electrocardiogram, which will lead us to operate earlier on these patients.

There is some disagreement whether or not to operate on patients with heart wounds. Many factors must be taken into consideration, as was brought out. In cases of disruption of the major coronary vessels or disruption of the conduction system, as frequently occurs in gunshot wounds, the patient will not live to get to the hospital. That leaves two preventible causes of death from wounds of the heart. One is tamponade and the

other is massive hemorrhage into the pleura or outside.

In a city where transportation of the injured to the hospital is fast, you will see patients who die from hemorrhage into the pleura or outside. In the city where transportation is slow, this will not be the case. You will see only the patients who will die from tamponade if untreated.

The difference between tamponade and death from hemorrhage depends upon relative flow capacities of the wound in the pericardium and the wound in the heart.

If the wound of the pericardium is sufficiently large to decompress the heart completely, you are likely to have death from hemorrhage. We do see such cases of death from massive hemorrhage into the pleura more so than to the outside.

In Louisville we still prefer operative management so that we can see what type of wound is present and deal with it under direct vision. From that standpoint, we have saved a considerable number of patients who had not only wounds of the heart but also of other structures such as internal mammary vessels and intercostal vessels. Those patients can bleed to death from intermammary and intercostal vessels as well as they can from other vessels.

James C. Drye (closing): We must remember that we are not talking about all patients with heart wounds, but rather those who get to the hospital alive. As Dr. Griswold brought out, the better the transportation, the poorer the patient because they are still alive upon arrival. In our series we included any patient who entered the hospital with any evidence of life.

If you rule out those patients in whom lack of time prevented treatment, of course, your operative results are much better. For this reason it is sometimes meaningless to compare statistics related to life or death in cases of wounds of the heart treated by different methods. However, our immediate results, using routine pericardiotomy, compare favorably to those in which aspiration was the basic treatment.

Dr. Chamberlain's paper disturbs me. In our experience tamponade is a very real thing, at least in the human. We have seen many patients in profound shock, accompanied by venous distention, in whom we have found tense, blue pericardiums on opening the chest. As soon as the pericardium was opened, the heart immediately began to beat forcibly, and there was a marked rise in blood pressure. In most cases blood loss was not too much of a problem. There was frequently some minor bleeding; this was controlled by placing the finger over the myocardial defect which was sutured. I think that suture does something more than merely stopping the blood. If left unsutured, these wounds may tend to separate and heal with only a thin layer of fibrous tissue. The result is a false aneurysm, as was suggested by Rudolph Matas in 1909. The main point of our paper is the fact that we found no evidence of heart disease due to the injuries to the heart, and, in particular, no evidence of constrictive pericarditis, a condition which has been reported following treatment by aspiration.

Charles G. Johnston (Detroit, Mich.): In relation to Dr. Drye's paper, I would like to say that we too have carried out such a study, which I believe was published some years ago by Dr. Smathers.* We were not able to find early evidences of pericarditis by electrocardiogram. Patients were studied for six or eight years after injury with no evidence of any particular heart disease. So I assumed that once these patients got well, they were well.

I am sure that if, as Dr. Avery said, Dr. Chamberlain has saved many lives of animals by presenting the assumption that there is no such thing as tamponade, and if this becomes generally accepted by surgeons who treat such patients, the author will not be doing very much for humans. I say this because whether or not tamponade occurs in the dog, it certainly does occur in the human. The assumption that all the blood will run out if there is a hole in the pericardium is not true because occasionally we do not see blood leaking out when the chest is opened, only a slow drip of blood from a tense pericardium. If there is no such thing as pericardial tamponade, and I am sure there is because I have seen it, why does any one aspirate the pericardium in these cases? This is a popular form of treatment.

I agree with Dr. Griswold's view for we also favor operation upon these people, again because it is associated with a major blood supply and we do not know what the damage may be.

I have seen one patient who, two days after tamponade was relieved by aspiration, blew out a clot from the wound and promptly died. When patients have lost blood and have been in some difficulty for two days, and then massively lose another large amount of blood, the damage is quite serious. Therefore, I am not particularly in favor of aspiration, although occasionally we do temporize by aspiration. I would much rather know what the damage is; and since these patients do well after operation and the mortality is not too high, I prefer exploration. If you can close the wound in the heart, it is much better. The fact has been shown in these cases that clots do form very fast, but one must remember that when a clot forms in a fairly good-sized opening, the clot can dislodge and when it dislodges, it is likely to be serious.

*(*Am. J. Med. Sc.*, 218: 213–224, 1949.)

I am a little chagrined about the fact that when Dr. Johnson came to me to discuss this case I had never seen nor heard of one. He asked if he could look through the cases we have had, and he found one such case which was pretty well identified in our own series and about which I had not known.

I think this is a rather forward step, for while these cases are very rare, they are interesting. Dr. Johnson has not only discovered it but has also described the symptomatology and electrocardiographic findings well enough to suggest that the patient had a ruptured pericardium. However, it is difficult for me to understand how a blow to the pericardium will allow the heart to escape without untold damage.

ARAN S. JOHNSON (closing): The only concluding remark I wish to make is in the form of a question. In instances of sudden death in individuals who have sustained a severe blow to the chest, principally professional fighters, might the cause be the result of partial fracture of the pericardium with incarceration of the heart? We must be aware of this condition since immediate exploratory thoracotomy may prove to be life-saving.

J. MAXWELL CHAMBERLAIN (closing): I am sorry if I have left the impression that I do not believe in tamponade. Actually I wanted to divert the emphasis from tamponade to circulatory collapse, and point out the basic principle that myocardial anoxia and shock are extremely important factors in the cause of death.

In the literature, as reviewed, we have noticed that at operation the surgeon often finds either no bleeding or an insignificant amount of bleeding from the injury to the myocardium. The point I wish to make, however, is that the suture used to stop bleeding from a branch of the coronary system in no way improves the nutrition to the myocardium distal to that ligature.

My plea is for restoration of nutrition to the myocardium to avoid myocardial anoxia. Dr. Cassebaum has called attention to the patient with coronary thrombosis. Such patients survive because of intercoronary collateral circulation—a gradual development in coronary sclerosis. Stab wounds of the heart are acute episodes in the life of the patient and the circulation is interrupted suddenly for the first time.

I believe in tamponade. I am sure it exists, just as everybody else does; otherwise, I would have to deny all the good work that has been done in the past by Drs. Griswold and Johnston and others. We have gone into the details of tamponade in our paper.

I should like to agree with Dr. Griswold that hemorrhage is one of the important factors leading to shock and death. Such patients can lose tremendous amounts of blood into the pleural cavity. The shock and the importance of the duration of the shock, with its irreversible aspects, are also discussed in the manuscript.

Dr. Drye's discussion has been most helpful to me. Pericarditis did not develop in any of his operated patients. In the movie,* when I removed the clot in the pericardium, I was sure someone would say, "Well, if for no other reason, you ought to operate to remove that clot because it is going to cause constrictive pericarditis." Actually in the literature constrictive pericarditis remains an inconspicuous complication.

I am sure that if Drs. Cassebaum, Griswold, Johnston, Drye, Avery and I all saw the same patient at the same time and under the same circumstances, we would probably all come to the same conclusion.

* Dr. Chamberlain illustrated his paper with a brief motion picture on the basic problems in the management of stab wounds of the heart.

Surgical Treatment of Acquired Arteriovenous Fistulas*

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FOR many years quadruple ligation and excision has been the method of surgical treatment of acquired arteriovenous communication. Recent investigations,[9] particularly in centers caring for war casualties, suggest a reduced incidence of arterial insufficiency when arterial continuity is re-established by end-to-end anastomosis, homologous artery graft or autogenous vein graft. Figure 1 illustrates a situation found frequently in our series.

Stimulated by these recent innovations in vascular surgery, we have undertaken a study of thirty-two patients afflicted with traumatic arteriovenous fistula of a limb; the treatment in each had consisted of interruption of the arterial and venous circulation in the limb by means of ligation and excision of the involved vessels. Surgical specimens were submitted to gross and microscopic examination; questionnaires sent to patients afforded the follow-up evaluation of each limb treated by ligation and excision. Particular emphasis was placed on adequacy of the interrupted arterial and venous circulation.

Although much has been written from early times about the arteriovenous fistula, it remained for William Hunter in 1759 to distinguish it from a simple aneurysm; he was the first to give a detailed description of the signs, symptoms and physical findings of the affliction. The major clinical manifestations are a harsh thrill and a bruit in the involved area; these are continuous throughout the cardiac cycle with systolic accentuation. The rapid passage of arterial blood into the venous circulation may result in marked elevation of the venous pressure and thence in chronic venous insufficiency with varices, edema and stasis pigmentation; also, one sees ulceration of the limb. Figure 2 shows the increased venous pattern and generalized enlargement of the limb with a long-standing fistula. Another frequent finding is cardiac enlargement.

One may make a diagnosis of acquired arteriovenous fistula when a thrill and a bruit are observed in the region of a penetrating wound; the presence of chronic venous insufficiency and increased warmth in such a limb in the region of the fistula is further evidence of an arteriovenous shunt. In our experience arteriography was not necessary to enable one to establish the diagnosis.

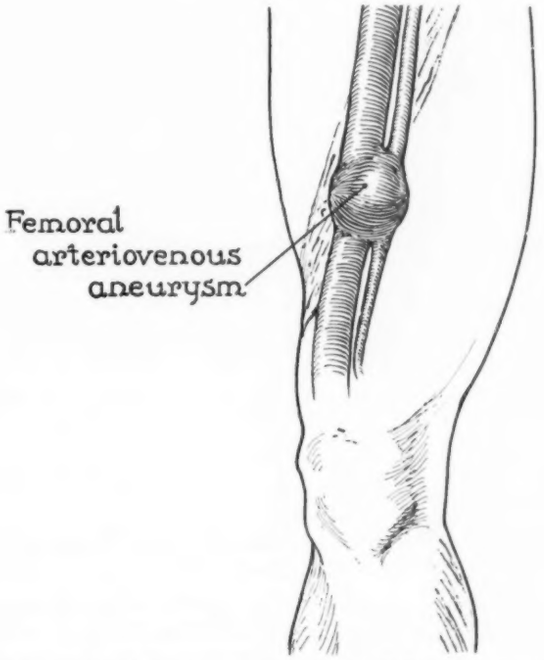

FIG. 1. Arteriovenous aneurysm involving the superficial femoral artery and vein.

* Abridgment of portion of thesis submitted by Dr. Foley to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Orthopedic Surgery.

FIG. 2. Infrared photograph showing fistula of forty-two years' duration. Note increase in size of right leg and venous pattern depicted.

A study of the profuse literature on the subject shows that our present knowledge has resulted from the contributions of many authors for the past 200 years. Holman[5] has pointed out that it was William Hunter who first noted that the bruit and the thrill are stopped by digital pressure applied to the fistula or the proximal part of the artery. Breschet found that gangrene results when only the proximal segment of the artery is ligated. Some success has been achieved when both the proximal and distal portions of the arteries are ligated. It is the problem of recurrence of the arteriovenous shunt that is the disadvantage of operations other than those consisting of complete excision of the fistula and accompanying vessels.

In 1886 Bramann condemned ligation of the proximal segment of the artery and suggested quadruple ligation of the artery and vein, and extirpation of the communication. Makins extended the use of this operation early in World War I.

Transvenous closure of an arteriovenous fistula was first suggested by Bickham in 1904. This early attempt at preservation of the arterial continuity was based on the Matas technic of intravascular repair. Such a procedure may be employed in recently acquired fistulas in which no degenerative changes exist in the vessel walls; however, in fistulas of long duration it is often impossible to separate the vessels. In such cases the surgeon is forced to remove the involved arteries and veins. Subsequent formation of an aneurysm at the site of arterial degeneration has been recorded following transvenous repair.

Experience has shown that proximal ligation of the artery and vein does not eradicate the arteriovenous shunt because collateral vessels carry the blood around the site of ligation.

In a further attempt to develop a safe treatment for acquired arteriovenous fistula, some surgeons have ligated the artery and restored the vein. The frequent occurrence of gangrene discouraged further use of this procedure. It is reasoned that what small amount of blood passes the ligated area goes quickly into a dilated venous system without entering the distal circulation.

Studies have shown that the incidence of gangrene is significantly reduced when ligation of the satellite vein accompanies ligation of the artery. This finding is based physiologically on studies by van Kend, who demonstrated a fall of blood pressure in a limb on ligation of an artery, followed by a slight rise on ligation of the accompanying vein.

The majority of authors have agreed with Holman,[6] who until recently has upheld quadruple ligation and excision of the fistula as the safest and most effective method of dealing with acquired arteriovenous fistula in a limb. With this operation no collateral channels will

open to restore the local bruit, and the danger of gangrene is not grave because of the good collateral circulation to the distal portion of the limb. Ligation of the artery and the vein is done with heavy silk sutures a few centimeters proximal and distal to the fistula. The H-shaped segment is then excised and a large calcified aneurysmal sac often is seen. (Fig. 3.) Vascular insufficiency may result when collateral circulation is poor in the involved limb; gangrene also may occur. A sympathetic block or resection may be necessary either before or after such an obliterative operation; indications for sympathectomy are intermittent claudication, pallor on elevation of the limb, dependent rubor and delayed venous filling time. It often happens that multiple vessels are concerned in a fistula. Each of these, of course, must be ligated. Hence, more often than not the operation consists of multiple ligation and excision rather than quadruple ligation and excision.

TABLE 1
EFFECTS OF LIGATION AND EXCISION ON ARTERIAL AND VENOUS CIRCULATION

No. of cases followed up	24
Upper extremity; no arterial or venous insufficiency	4
Lower extremity, total	20
Arterial insufficiency (10)	
Mild (7)	
Moderate (1)	
Severe (2)	
No arterial insufficiency (10)	
Venous insufficiency (15)	
Mild (7)	
Moderate (6)	
Severe (2)	
No venous insufficiency (5)	

During the past ten years prominent investigators[4,6] have advocated restoring a pulsating arterial blood flow for complete restitution of function in legs with damaged or diseased primary arteries. This must be done without damaging collateral vessels.

Good follow-up studies are available on twenty-four of the thirty-two patients concerned. (Table 1.) Of these patients, twenty had fistulas of a lower limb and four had involvement of an upper limb. There was no postoperative arterial or venous insufficiency in those patients operated upon for arteriovenous fistula of an upper limb. All four had some impaired use of the involved arm. All twenty-four patients in the follow-up study were in good health.

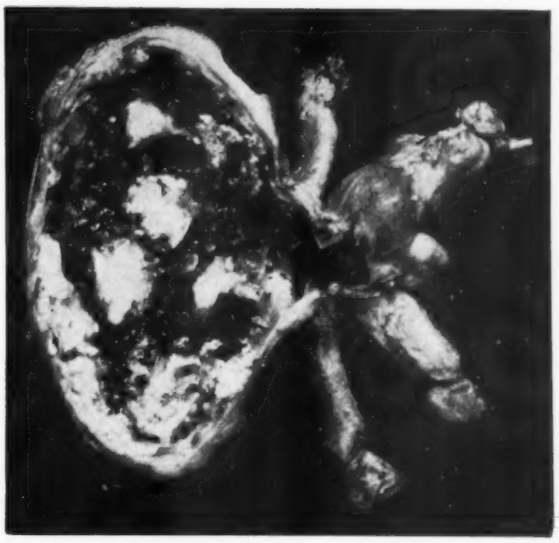

FIG. 3. Calcified arteriovenous aneurysm of superficial femoral vessels of nineteen years' duration. Note the large calcified aneurysmal sac.

Twenty patients underwent surgical interruption of vascular continuity in their lower limbs; ten had residual arterial insufficiency, whereas an equal number had good circulation to the involved limb. The arterial insufficiency was judged to be mild in seven, moderate in one and severe in two. We were unable to predict in which patients arterial insufficiency would develop after quadruple ligation and excision. The state of the peripheral pulses and the location of the fistula did not correlate in any way with the presence of arterial insufficiency after ligation and excision of the arteriovenous fistula. On the other hand, venous insufficiency usually persisted after operation but was not seen to begin after surgical correction of the fistula.

Fifteen of the twenty patients had some venous insufficiency. This was graded as mild in seven, moderate in six and severe in two. It appears that the longer an arteriovenous fistula persists the more severe is the disability from venous insufficiency. The fact that this venous insufficiency persists after operation and is the major source of long-term disability suggests that early correction of the fistula is advisable.

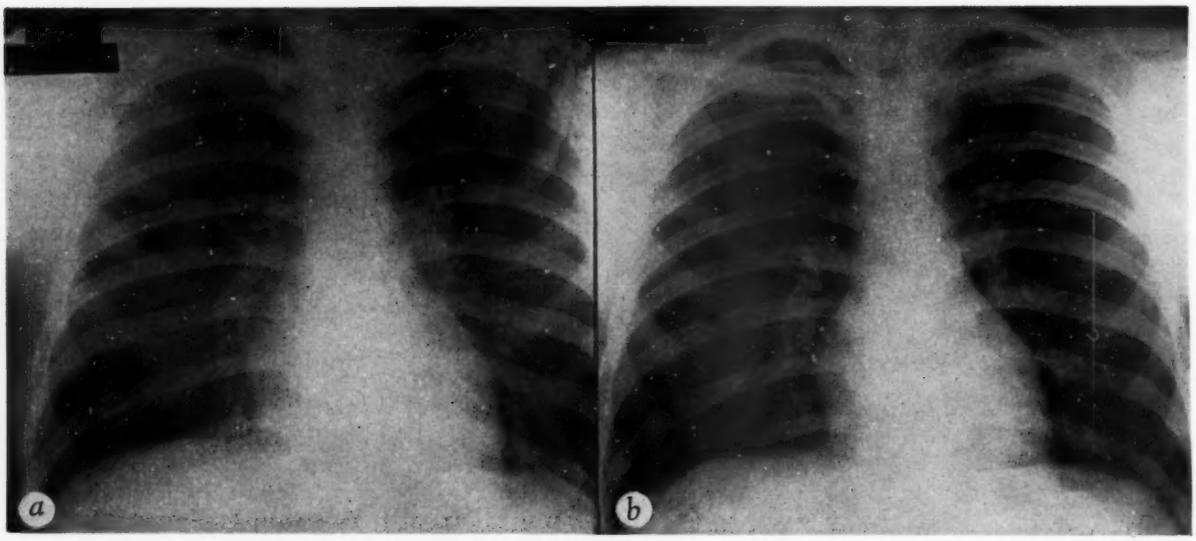

FIG. 4. (*a*, *b*) Roentgenograms show decrease in size of the heart two weeks after quadruple ligation and excision.

There was no correlation between the presence of arterial and venous insufficiency except in those two patients in whom both arterial and venous insufficiency were severe. The fistulas often were of long duration. The shortest period of arteriovenous shunting was five weeks; the longest was forty-two years. The average duration of a fistula was 13.5 years; this period far exceeds that of fresh lesions seen at centers of the armed forces.

Electrocardiograms were reviewed with the help of Dr. H. B. Burchell. It is worthy of note that all tracings were essentially normal. The heart rate never exceeded 100 beats per minute. High voltage R and T waves were encountered in left precordial leads; this finding did not correlate with that of increased size of the heart as determined by roentgenograms. The electrocardiographic findings suggested left ventricular hypertrophy.

The oldest patient in our series was fifty-nine years of age; the youngest, and only female, was nine years. The average age of the patients was thirty-six years.

The cause of the fistula in the thirty-two patients was gunshot wounds in twenty-five, stab wounds in five and flying metal in two.

Examiners were more impressed with complaints and findings referable to venous insufficiency than with indications of arterial insufficiency. There was gangrene of distal parts in only one patient, and this involved only the toes.

The classic pattern of symptoms and signs was common among the patients in our series. Often seen were venous stasis, edema of the involved leg, a pulsating mass, bradycardia on compression of the fistula and increased circumference of the involved leg. Ankle or pretibial ulceration was present in a third of the patients.

There was preoperative enlargement of the heart in half the patients. The earliest postoperative roentgenogram of the chest was taken two weeks after operation. (Fig. 4.) The heart had returned to normal size; obliteration of the arteriovenous shunt had the same effect on the size of the heart whenever it was found to be enlarged before operation.

In five patients there was increased length of the affected leg; all such patients had been injured in adolescence. Increases in length of 1 inch or less were noted.

Good preoperative collateral circulation was present in all but one of the surgical candidates. This was corroborated at operation by a positive Henle-Coenen sign; that is, good pulsation and blood flow were present in the distal arterial segment when the proximal portion of the artery was occluded. Compression of the proximal portion of the artery stopped the bruit in all patients on whom it was tried.

Fourteen patients had an arteriovenous fistula involving the superficial femoral artery and vein. (Table II.) This was the most common site. The remainder of the patients had fistulas scattered among other vessels of the limb.

In addition to ligation and excision of the fistula and accompanying vessels there were four instances in which interruption of the sympathetic innervation was necessary. One patient underwent cervicothoracic block on the day of operation. In a patient who had undergone ligation of the superficial and deep femoral vessels, numbness of the toes and pallor of the foot present on the fifth postoperative day indicated alcoholic sympathetic block. A third patient, who had undergone excision of the popliteal artery and vein, was given a postoperative sympathetic block because of coldness of the distal portion of the limb. This warmed the limb. In one patient it was necessary to perform preoperative lumbar sympathectomy because of obliteration of distal pulses when the popliteal arteriovenous fistula was compressed. Two weeks later a fistula of the posterior tibial artery and vein was removed. Follow-up study revealed an excellent result in this patient.

TABLE II
ARTERIES AND ACCOMPANYING VEINS INVOLVED

Superficial femoral	14 (44%)
Common femoral	4
Posterior tibial	4
Popliteal	3
Subclavian	2
Radial	1
External iliac	1
Profunda femoris	1
Brachial	1
Axillary	1

An arteriogram was taken in five instances. (Fig. 5.) The fistula was well displayed in each case; that is, a good picture of iodopyracet (diodrast®) moving from the arterial to the venous circulation was seen. In Figure 5 one sees an enormous aneurysm of the proximal segment of the vein in the pelvis. A similar aneurysm in another patient resulted in marked displacement of the bladder. These tests were done with about 30 cc. of 35 per cent solution of diodrast.

There were postoperative complications in ten patients. These are shown in Table III.

One of the more interesting phases of our study was an examination of the gross and microscopic appearance of the blood vessels. Here we wish to thank Dr. D. C. Dahlin for his aid in the work with surgical specimens. An appreciation of alterations in structure was heightened by comparison with normal control vessels. The diameter of the fistulas ranged

FIG. 5. Femoral arteriogram. Note arteriovenous fistula in mid-thigh and enormous aneurysm of proximal segment of the vein in the pelvis. The aneurysm was excised when the fistula was removed.

TABLE III
POSTOPERATIVE COMPLICATIONS IN TEN OF THIRTY-TWO CASES

Pulmonary embolism	2*
Wound hematoma	4
No gangrene (3)	
Gangrene, mid-thigh, amputation required (1)	
Thrombophlebitis	2
Lymphadenitis and lymphangitis	1
Weakness of calf muscles	1
Recurrence with thrombosis of profunda artery (3 yr. postoperative period)	1

* One death.

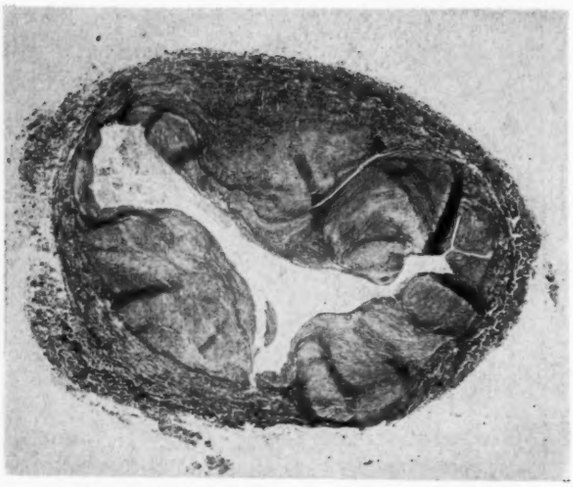

FIG. 6. Marked intimal fibrosis of vein. Fistula present for nineteen years (elastic van Gieson; original magnification × 5½).

from 3 to 20 mm.; the average diameter was about 7 mm.

Examination of preserved arteries and veins revealed some variation in size of the lumen and thickness of the wall. The most common picture consisted of a normal distal segment of the artery and an enlarged proximal segment of the artery; this discrepancy in the size of the proximal and distal segments of the artery makes it difficult to place a graft spanning the defect. The proximal and distal segments of the vein always were enlarged considerably. A thickening of both the proximal and distal walls of the vein was seen; this is the so-called arterialization of veins.

All vessels were examined microscopically after they had been stained with hematoxylin and eosin for cellular detail and with elastic van Gieson's stain for stromal pattern. As in the gross specimens, the distal segments of the arteries so studied were normal. Generalized hypertrophy of all layers explained the thickening of the proximal arterial wall. The thickening of the wall of the vein was due to intimal fibrosis (Fig. 6); this could be graded 1 to 4, based on the thickness of the fibrous tissue deposits. This intimal fibrosis is similar to the jet lesion of patent ductus arteriosus. We expected to find the media of the vein thickened to resemble that of an artery; however, the media was essentially normal. Elastic fibers were found frequently in the intima; at times even a distinct internal elastic lamina was noted on the edge of the lumen.

Microscopic examination of the fistula and the aneurysmal sac showed these to be composed of a hyalinized fibrous wall. Sometimes calcification of the wall was also seen.

Our studies revealed definite correlation between the severity of the intimal fibrosis of the veins and the duration of the fistula; that is to say, there usually was low grade intimal thickening in fistulas of two years' duration or less, and grade 3 to 4 fibrosis in the presence of fistulas of longer duration. To our surprise it was uncommon to find degenerative changes in the arteries and veins. There was thrombosis in the proximal segment of one artery; calcification was seen in the distal portion of another artery and the distal portion of one vein was calcified. We infer from this study that most arteries and veins involved in long-standing arteriovenous fistulas are in a healthy state.

COMMENT

This is a study of thirty-two patients with long-standing traumatic arteriovenous fistulas of the limbs. It is evident that the fistula is well tolerated but that it causes symptoms of chronic venous insufficiency.

All patients were treated with quadruple ligation and excision; this was well tolerated in the four instances in the upper limbs. However, some impairment of function results from interruption of the major arteries and veins in the lower limbs. Twenty-three of the twenty-four patients in the follow-up studies are able to work at gainful employment. Fifty per cent of our patients with interruption of arterial continuity in the lower limbs experienced postoperative arterial insufficiency; in most instances this limited only strenuous physical activity.

We are unable to predict in which patients arterial insufficiency will develop after quadruple ligation and excision. On the other hand, as would be expected, venous insufficiency usually persists after correction of the arteriovenous fistula; 75 per cent of the patients had postoperative venous insufficiency after ligation and excision of a fistula in a lower limb.

A plea is made for the early surgical treatment of traumatic arteriovenous fistulas. It is hoped thereby to avert the chronic venous insufficiency which plagues these patients.

The surgeon should attempt, when possible, to re-establish the arterial and venous continuity in the lower limb when treating trau-

matic arteriovenous fistula. If this cannot be done, satisfactory results may be obtained in most instances with quadruple ligation and excision.

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DISCUSSION

Joseph M. Janes (closing): Traumatic arteriovenous fistulas are not often encountered in civilian practice. Most large series of this condition have been reported from the armed services.

For many years we have thought the quadruple ligation—excision operation was an adequate one; but when the surgeons in Korea began to advise restoration of the continuity of the artery, and when Holman, who for many years has advocated quadruple ligation and excision, began to advise restoration of the continuity of the artery, we thought it was time that we looked into some of our cases to see the results of the treatment.

Dr. Foley has presented the summary of our investigation. I think there is one thing that we have to bear in mind. Whereas the experience of the army and navy surgeons in these conditions is with the recent case, cases reported in our study average 13.5 years in duration. Although it is probably desirable to reconstruct the artery and if technically feasible to reconstruct the vein, I think there will always be cases in which the quadruple ligation-excision operation will be performed.

Since 1950, in selected cases of children with a short lower extremity, we have been creating an arteriovenous fistula in the femoral vessels; to date we have done seventeen. Six of them have come to closures for one reason or another. In two of them we performed quadruple ligation and excision, and in two we did a transvenous repair of the artery.

It so happens in these four cases that the two patients who had the transvenous repair had less arterial circulation in their foot on plethysmograms than those who underwent quadruple ligation and excision.

However, from four cases we cannot say too much. It is our hope that as we close these fistulas we may do a series of transvenous closures and a series of quadruple ligation and then take plethysmograms to see which procedure gives the more adequate circulation distally.

The Fractured Hip—A Major Surgical and Sociologic Problem

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THE fractured hip is a problem of ever increasing importance as the geriatric case load throughout the country rises. Over the course of the past ten years slightly over 500 patients with fractured hips have been treated at the Edward J. Meyer Memorial Hospital, Buffalo, New York. Rather than considering the amassed data from these cases, we have chosen instead to present two series, each consisting of over 100 patients. The first series includes consecutive cases treated very early in the period, while the second series represents consecutive cases treated more recently. These two series, with about a five-year interval between them, are then available for comparison: series A (1946–1948), 120 patients; series B (1953–1954), 119 patients. The patients in both series were predominantly treated by the resident staff with the close supervision of the general surgical and orthopedic attending staffs.

In this report we have limited ourselves to consideration of survival rather than of fracture union, since survival is obviously of paramount importance in treating any disease. Reversing the usual practice in which over-all mortality figures are presented last, we would like to present them first. Thus our over-all hospital mortality is: series A (1946–1948), 36.7 per cent, series B (1953–1954), 27.8 per cent.

These figures would appear to indicate an approximate 9 per cent reduction in mortality of the later group. Since the pitfalls of loose interpretation of clinical data had recently been re-emphasized by Gibbon,[1] we believed it imperative that our data be submitted for statistical analysis of possible significance. At the hazard of insulting the reader's intelligence, it is believed that the probability of a certain configuration of data being due to chance should be less than one in twenty before the data can be considered significant with a relative degree of certainty. Our mortality data is not statistically significant when subjected to such analysis. From a statistical standpoint, our mortality is unchanged over the period of study. We are continuing to lose about one-third of all patients admitted with a fractured hip, a rather appalling mortality.

An attempt to compare this mortality with that reported by others led us into increasing confusion, as the data in Table I will indicate.

Although we freely admit the material selected for this table was chosen to illustrate a point, it certainly should be apparent that reported mortality rate alone may be extremely misleading. The element of case selection becomes of paramount importance, so that comparisons of series reported by different authors become meaningless.

It having become apparent that comparison with other series was rather fruitless, we concentrated on determining, if possible, what differences existed between our two study groups and why we were unable to show the significant decrease in mortality, which our ego had led us to expect.

Table II shows the average ages for the survivors and the deaths in the two groups.

While average age has been the single most

TABLE I
SELECTED REPORTED MORTALITY RATES

Author	No. of Cases	Mortality (%)
Lofberg (1927)[2]	264	6
Conwell (1939)[2]	1,485	8.5
Cleveland (1951)[3]	307	16
Montsch (1952)[4]	250	27
Present series	.....	32

constant element of selection apparent in correlating different series with their mortality rates, our case material is identical as to age in the two groups. As might be expected, there is a significant age difference between the survivors and the deaths.

TABLE II
AVERAGE AGE

	Survivors	Deaths
Series A	72 yr.	79 yr.
Series B	73 yr.	78 yr.

Eighty-five per cent of the patients of both series had serious coexisting disease at the time of admission, of which cardiovascular and pulmonary diseases were predominant.

One-quarter of the patients in each series sustained the fracture in an institution: a hospital, nursing home or old folks home.

Insofar as it is possible to gauge the similarity of case material in two groups, these series would seem to be comparable.

Regarding method of treatment, the percentage of the total cases which were operated upon in the two groups, along with the surgical mortality, is shown in Table III.

The marked increase in the number of cases operated upon without increasing the surgical mortality would seem to be a very significant trend. This undoubtedly results in a shorter hospital stay and higher incidence of bony union; but, as mentioned, in this series it has not produced a statistically significant reduction in over-all mortality. The report of Scott[5] is of considerable interest in this connection. He studied a group of fifty-four consecutive patients with intertrochanteric fractures by a strictly alternate case method: one being operated upon and the next being treated by non-operative methods. There was no statistical difference between the mortality rates in the two groups. Although we would like to agree with Evans[6] that, "The evidence in support of the claim for a lowered mortality among patients treated by operation is overwhelming," there also appears to be a large volume of data indicating the reverse.

We do not treat the fractured hip as an emergency. The average interval from admission to operation is 5.4 days in both series A and B.

This rather long interval between admission and operation has frequently been the subject of controversy in our local trauma conferences, since it obviously violates the usual principles

TABLE III
PERCENTAGE OF CASES OPERATED AND SURGICAL MORTALITY

	Total Operated upon (%)	Surgical Mortality (%)
Series A (1946–1948)	56.5	26.5
Series B (1953–1954)	80.0	25.6

of fracture management. For a brief period these cases were handled as surgical emergencies with internal fixation being performed immediately on admission. The mortality rate increased markedly while the technical quality of the fixation decreased sharply, so that the procedure was soon abandoned. The best in anesthesia, nursing help and radiographic technic are necessary to obtain the lowest mortality and highest quality of technical fixation, neither of which we were able to obtain in the middle of the night. Although this interval from injury to operation is omitted in many reports, Cleveland and Fielding[7] report an average interval of 4.2 days for their service cases.

Table IV shows the change in anesthesia trends at our institution. We are unable to say

TABLE IV
TYPES OF ANESTHESIA USED

	General (%)	Spinal (%)	Local (%)
Series A (1946–1948)	69	25	6
Series B (1953–1954)	20	80	..

whether the striking increase in the use of spinal anesthesia has altered the outcome.

The average operating time for the separate years from which the cases were studied are: 1947, 1.6 hours; 1948, 1.7 hours; 1953, 2.0 hours; 1954, 2.8 hours. We cannot state whether this indicates that we are taking more

time in order to do a better job or are just getting slower.

The enormous economic problem created by these cases is apparent from Table v. This shows the total hospital days required by the patients in each group, along with the cost of that hospitalization.

TABLE V
TOTAL HOSPITALIZATION AND ITS COST

	No. of Cases	Total Hospital Days	Charge per Day	Total Cost of Hospitalization
Series A (1946–1948) . .	120	7,375	$10.00	$73,750.00
Series B (1953–1954) . .	119	5,552	$15.50	$86,000.00

While the average number of hospital days has dropped from sixty-two in the early series to forty-seven in the later series, the average cost of hospitalization has increased from $620 per case to $720 per case due to the increase in hospital rates. Under the present insurance plans a negligible proportion of this cost is defrayed by insurance since most of the patients are too old to be eligible for such insurance.

Another point to be considered is the influence of total hospital days on mortality rates. As has been shown by several reports in which hospital mortality and three to six month-mortality rates were stated,[5,8] over-all mortality continues to rise with the length of time the patients are observed.

A county institution such as ours, having an average hospital stay of two months, can be expected to have an appreciably higher hospital mortality than a private hospital where the average stay is three weeks. The numerical difference in our mortality rates might be due to the shorter hospital stay in the later series.

SUMMARY AND CONCLUSIONS

1. The fractured hip case load is increasing annually.

2. The over-all mortality rate has not changed significantly in the past ten years in this institution, in spite of a sharp rise in the percentage of cases treated by internal fixation.

3. The average cost of hospitalization per patient continues to rise.

4. There is no justification for an attitude of complacency toward the problem of the fractured hip.

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Rigid Internal Fixation of Intracapsular Femoral Neck Fractures

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SINCE the close of World War II the Jewett-Wright Orthopaedic Clinic has treated 554 fractured hips. Of these, 244 or 44 per cent were subcapital or transcervical fractures. The first fifty-six cases were treated with a Smith-Petersen nail in 1946 and early 1947, and are excluded from this series. Of the remaining 188 cases, 124 have been followed up for six months or longer. The average length of follow-up is twenty-two months. The minimum follow-up is six months, and the longest follow-up is seven and a half years. All of these cases were initially treated with internal fixation using the author's nail, and in no instance was the hip joint opened. The approach was from the upper lateral femoral incision, starting a little below the base of the greater trochanter and going distalward for from 5 to 7 inches. We believe that a few of these hips should have been opened up so that the reduction could have been made under direct vision, but in this series this was not done in any case. Of these 124 cases, 104 or 83.9 per cent obtained good bony union of the fracture. (Figs. 1 to 8.)

The results of our cases are classified in four categories: *excellent* if the patient is walking without any support and without pain and is well pleased with the result of his operation; *good* if the patient has some pain or limp and some need of occasional support; *fair* if the patient has union but pain persists and mild support is required; *poor* if the patient has non-union of the fracture with or without aseptic necrosis of the head of the femur. Of this series eighty-one or 65.3 per cent had excellent results. Fifteen or 12.2 per cent had good results. Eight or 6.5 per cent had fair results. Twenty or 16.1 per cent had poor results with non-union of the fracture. In eleven of these twenty cases the head and neck of the femur was replaced with a prosthesis.

We do not consider a fractured hip a surgical emergency. These patients are put in traction when they are admitted to the hospital. The routine laboratory procedures are carried out. If indicated the patient is checked by an internist.

At surgery the fracture is manipulated and reduced either by traction, abduction and internal rotation of the leg, or by Leadbetter's maneuver. Roentgenograms are made to confirm the satisfactory reduction of the fracture after a good Leadbetter test is obtained. We do not believe that the beak, which is sometimes found on the proximal fragment, necessarily prevents good reduction of the fracture. It was rarely necessary to open the hip joint to secure good reduction of the fracture. At surgery, after the femoral shaft was reached through a lateral approach, three guide wires were put into the neck and head of the femur, and checked by anteroposterior and lateral films. These guide wires should be directed toward the lower two-thirds of the articulating surface of the femoral head. Two guide wires are introduced into the head fragment above the wire to be used as a guide for the nail. This prevents rotation of the head fragment as the nail is driven in. It is not usually necessary to impact the fracture further. It is very important to have the tri-fin nail introduced as far as possible into the femoral head without entering into the hip joint, especially where there is a very shallow proximal head fragment.

As there is always absorption of bone at the fracture site, the end of the nail may penetrate through the head of the femur. In thirty-seven or 29.8 per cent of our cases the nail did protrude through the femoral head. Provided this is directed to the non-weightbearing portion or the inferior three-fifths of the acetabulum, no harm is done and rarely does the patient have any symptoms from this with the exception of some limitation of motion of the hip joint. In nine of these thirty-seven cases the protruding nail was replaced with a shorter nail since the fracture had not completely united at the time of this second operation. In six cases the nail

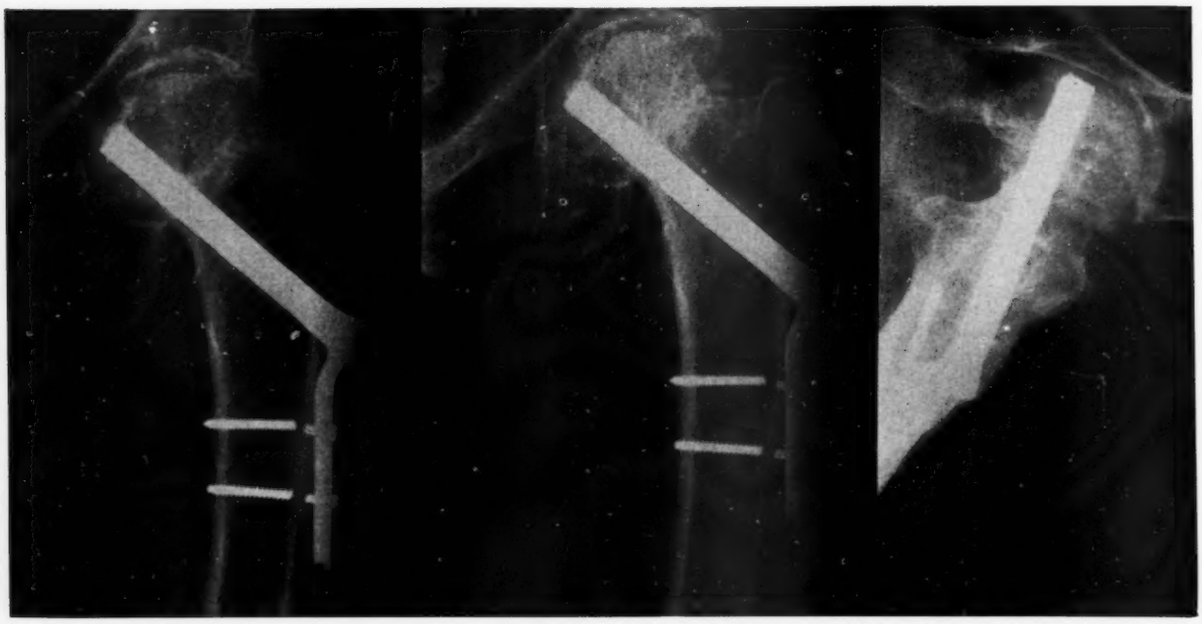

FIG. 1. Case I, J. S. These x-rays illustrate the strain put on the flange of the nail with breaking of the two screws. As described in one of my earlier articles, the crucial place to have strength and stability in any appliance for fractured hips is at the point of emergence of the nail at the base of the greater trochanter. Here is where the stress is transmitted down the shaft to be taken up by the screws, of which we now employ never any fewer than four and generally five or six. In this case, however, there was an excellent result.

FIG. 2. Case II, M. B. In this patient slight pain developed in the hip with some limited motion; at six months postoperatively the nail was removed and replaced with a shorter one. After this was done the pain disappeared and at thirty-four months' follow-up the patient had an excellent result. There were nine of these cases in which the original nail was replaced with a shorter one due to invasion of the hip joint by the first nail as absorption and healing took place. Most of these patients had excellent or good results.

FIG. 3. Case III, B. S. This patient had a slight pain in the hip and the nail was removed eleven months postoperatively. She had an excellent result at sixteen months' follow-up. There were six such cases in which the nail was removed due to protrusion through the femoral head.

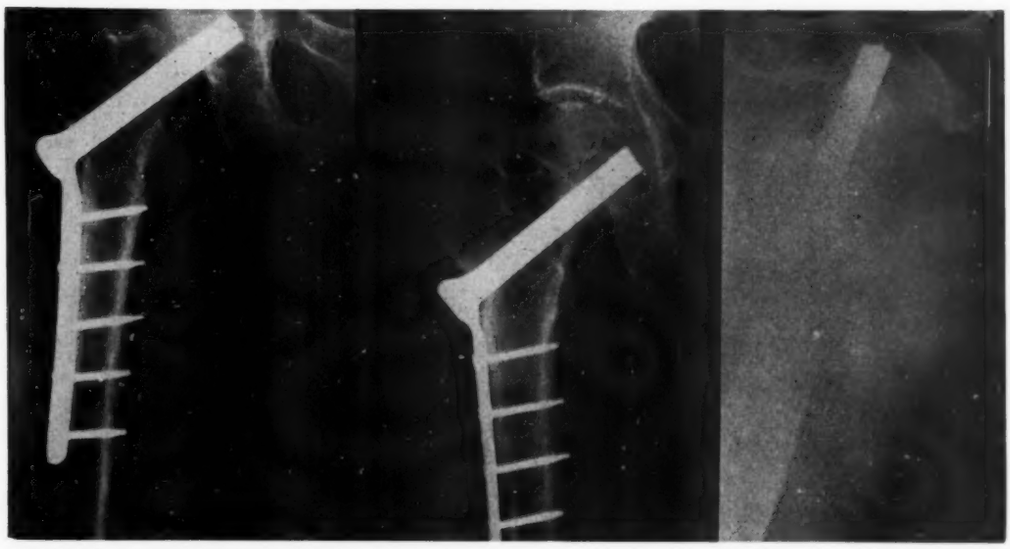

FIG. 4. Case IV, A. V. H. This patient's x-rays show a slight protrusion of the nail through the head without the patient having any symptoms. She had a normal range of motion and an excellent result at thirty-one months' follow-up. There were twenty-two cases in this category and in most of them there were excellent results.

FIG. 5. Case v, J. D. P. This patient's nail is slightly through the head and she was told repeatedly by one of the best known orthopedic men in the country that the nail would have to come out or the result would be a very bad hip joint; we disagreed with this. The patient continues to have no symptoms and has an excellent result approximately two and a half years postoperatively. She has a normal range of painless motion.

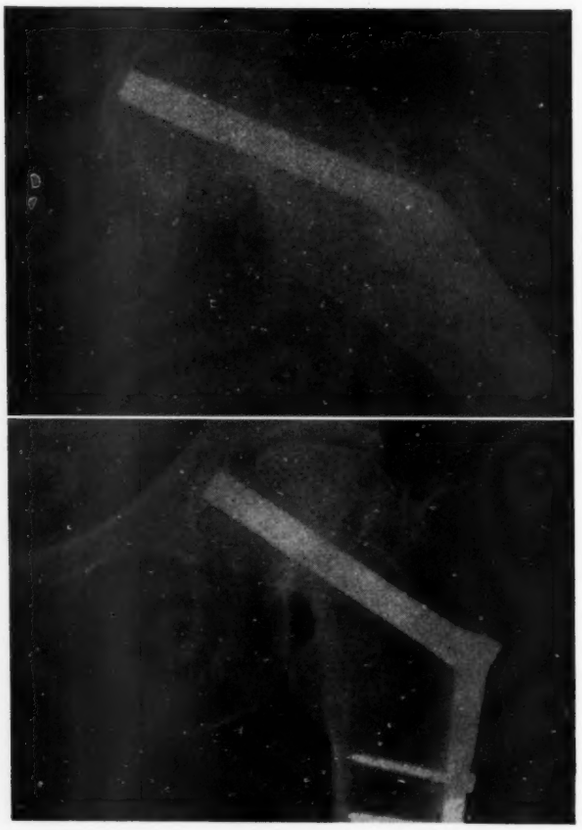

FIG. 6. Case vi, C. L. V. These x-ray show not only the protrusion of the nail without ensuing symptoms but also the head fragment inferior to the neck, which has repeatedly been said to be a definite cause of non-union. Also, the x-rays show that the "beak" does not have to be broken or osteotomized for good union. There was an excellent result on forty-four months' follow-up.

was removed after bony union was obtained. In twenty-two cases in which the nail protruded through the head of the femur the patients had no disturbing symptoms and refused to have the nail removed. Ambulation is insisted upon as soon as the general condition of the patient permits, which is usually within the first or second postoperative week. No or minimal weight-bearing is allowed, however, until there is definite evidence of bony union. In older people it is almost impossible to enforce complete non-weightbearing of that injured limb, and in order to encourage this the heel of the shoe on the good side may be raised ½ inch or so and the patient's foot on the injured side left covered only by a thin stocking. Patients do not want to walk on a good, sheer stocking although they would bear some weight if they had on a slipper or a shoe. Generally union is seen by x-ray within from three to six months, although sometimes it occurs even later.

Aseptic necrosis developed in thirteen or 10.5 per cent of the patients. Nine of these patients also had non-union of the fracture, but aseptic necrosis of the femoral head developed in four patients after good bony union of the fracture had been obtained.

The author has a hip prosthesis which has been employed now for approximately seven years in a series of forty-three cases. This prosthesis fits over the author's nail and has collars to regulate the distance of the greater trochanter from the upper acetabular brim. With the nail in place when the need for arthroplasty becomes evident, it is a relatively easy operation to enter the hip joint by any one of the three accepted incisions, remove the necrotic or non-united head, shape the neck accordingly and insert the collars and the prosthesis. Of course the hip then has to be reduced, putting the new head into the socket. Another paper will follow shortly dealing with the results of the use of this prosthesis but to date the results have been just as gratifying as

FIG. 7. Case VII, S. H. S. X-rays show that the beak need not be broken off for good reduction or fusion. They also show how the flange pulls away from the shaft of the femur but this nail did not have to be removed. Result was excellent at 33 months' follow-up.

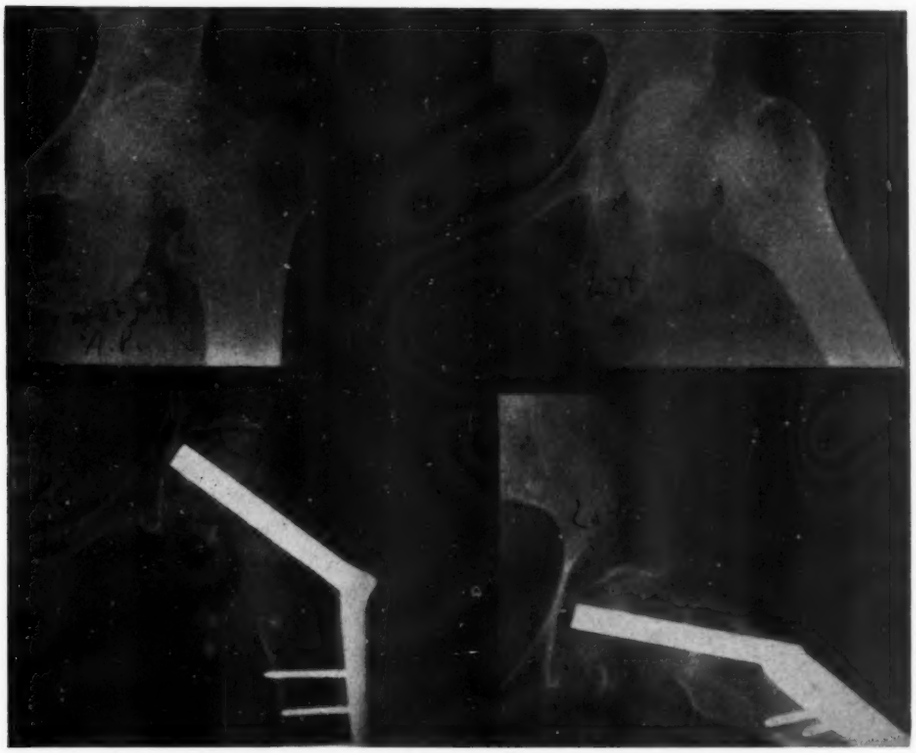

FIG. 8. Case VIII, E. H. X-rays again show intact beak which did not interfere with healing; also, protrusion of the nail which was not removed. At 34 months' follow-up, result was excellent with no pain.

any reported of the other prosthetic devices in common use.

CONCLUSION

We believe that the one-piece nail and plate affords firmer and better fixation of the fracture than any other known method. By the use of this nail we have obtained bony union in 83.9 per cent of the 124 consecutive cases in this series. In this series of 124 consecutive patients the average follow-up time was twenty-two months and none under six months postoperative are included. We have x-ray evidence of each and every case reported in this article and we have done everything in our power to make this analysis and report as accurate and factual as possible.

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Fracture-Separation of the Proximal Humeral Epiphysis

A STUDY OF CASES SEEN AT THE PRESBYTERIAN HOSPITAL FROM 1929–1953

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THIS study of forty-three cases has been carefully selected to represent a very specific type of injury, i.e., fracture-separation of the proximal humeral epiphysis. No injury to this epiphysis without visible x-ray evidence of separation is included in order to avoid confusion as to accuracy of the diagnosis. I have been unable to find in the literature a really adequate description of this injury accompanied with late x-ray follow-up and clinical evaluation of the patients. Originally it seemed to us very important to obtain correction of the badly displaced proximal humeral epiphyses, so much so that we made numerous attempts both by closed and open reduction to effect anatomic reduction and hold this with internal fixation if necessary. This present study extends over a period of twenty-five years, from 1929 to 1953.

The proximal extremity of the humerus develops from three centers of ossification, one for the head and one for each tuberosity. These unite when a person is approximately seven years of age and form a cap-like bony mass which rests upon the conical-shaped upper end of the shaft (diaphysis). On x-ray examination with the humerus internally rotated the epiphyseal line appears to cross in a transverse direction; with the humerus in external rotation the inner half of the epiphyseal line runs parallel to the anatomic neck from its medial border to the center, and then courses downward and lateralward to just below the greater tuberosity. Ossification becomes complete at about a person's eighteenth to twentieth year.

The common fracture-separation of the proximal humeral epiphysis is with the fracture line extending from the lateral side medially and then splitting off a variable-sized triangular-shaped fragment of the diaphysis, the same type of triangular diaphyseal fragment as seen in epiphyseal separations of the radius, lower tibia or lower femur.

From a review of x-ray films of these patients it becomes quickly evident how difficult it is to make a proper diagnosis, largely because of trouble in obtaining two-position views. The anteroposterior view in internal rotation (Figs. 1 and 2A) is readily obtainable, but to get a lateral or anteroposterior view in external rotation (Fig. 2B) is often impossible in the

FIG. 1. October 24, 1939. An anteroposterior view of the upper humerus in a five year old girl showing slight angulation of the shaft upon the epiphysis with a triangular diaphyseal fragment remaining attached to the epiphysis. The child was treated using sling and swathe for two weeks and made an excellent recovery without deformity.

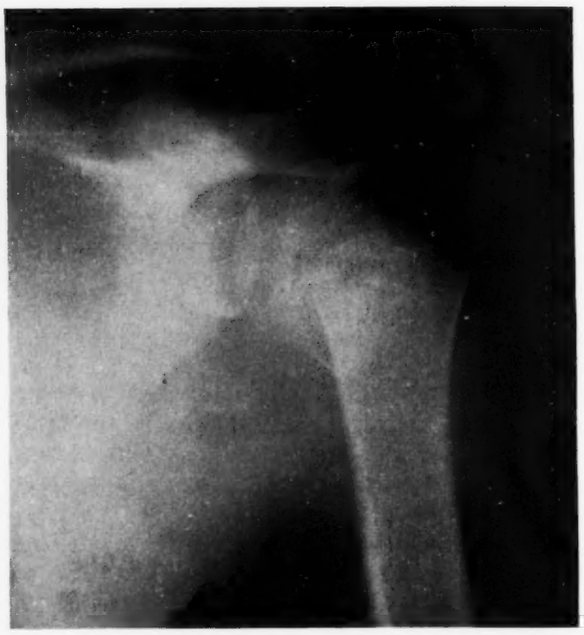


FIG. 2A. Patient was a sixteen year old boy. Anteroposterior view; arm in internal rotation showing mild deformity.

FIG. 2B. Same patient The arm is in external rotation and more marked deformity may be noted between the diaphysis and the epiphysis. This child was treated using sling and swathe for eight days and made an uneventful recovery; however, there was a 2.0 cm. shortening of the humerus.

early stages because of pain on positioning the patient's arm. An oblique or transthoracic view therefore *must* be made if the straight anteroposterior view fails to reveal displacement or angulation. (Figs. 3 and 6B.)

The common displacement of the distal fragment is in an upward, forward and inward direction due to the coracobrachialis and biceps muscle pull, whereas the proximal fragment rotates outward and forward. In several of our cases the upper end of the diaphysis has been shown projecting into the deltoid muscle; in one this end was originally caught in the deep surface of the skin, all but making this an open (compound) fracture. In some cases the lower fragment is shifted medially. Occasionally the fracture line runs the entire length of the epiphyseal line between cartilage plate and diaphysis without breaking off the triangular fragment usually seen. (Fig. 4.) The common type of displacement with head rotated outward and the shaft riding upward and inward is called the adduction type by most authors who describe a type, but it would seem to me to represent more closely the true varus type of fracture seen so commonly in femoral necks. The periosteum covering the upper diaphysis splits longitudinally to permit escape of this fragment and is displaced medially with the epiphyseal (head) fragment. (Fig. 5.) In children the periosteum is extremely tough; for this reason it often tightens and closes under the extruded shaft end on attempts to reduce the fracture and thus renders closed reduction difficult or impossible. If reduction is not achieved, new bone is then formed within the periosteum in the triangular space formed by the displaced epiphysis, the shaft and the periosteum itself. A bowing of the head and upper shaft thus results in the first few months of healing; however, with time, growth and physical activity this bowing becomes partially corrected and the originally displaced shaft becomes absorbed. (Fig. 8A to E.) Although we have known this for years, it was Aitken[1] who first recorded this fact in 1936.

In general most authors believe that where there is little or no displacement there need be no reduction. The patient's arm is kept quiet in a sling and swathe or Velpeau dressing for two weeks; progressive exercises and activity are then started.

Regarding moderate displacement or more, there are many suggestions in the literature as to the necessity for anatomic reduction, how to accomplish this and how to immobilize the arm after reduction. Key and Conwell[7] in their

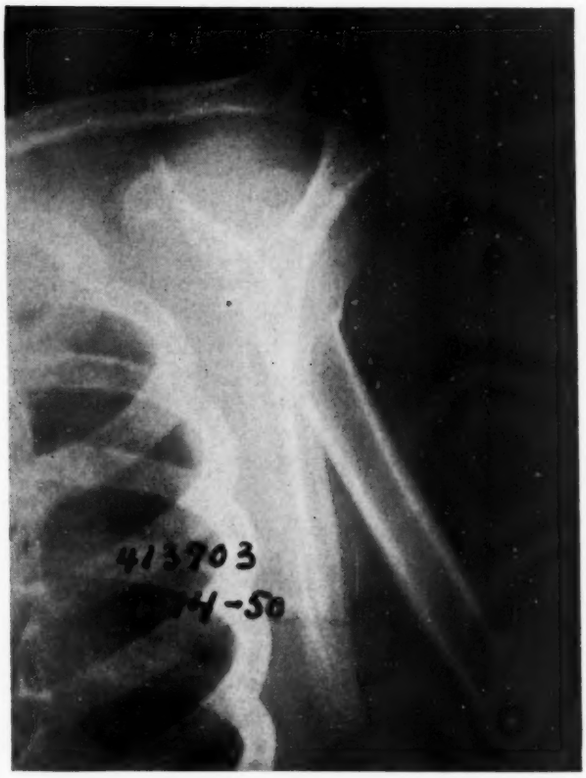


FIG. 3. This patient was a six year old girl. A tangential view of the upper humeral epiphysis showing slight separation which did not show in the regular antero-posterior view. The child was treated using sling and swathe for eleven days and made an excellent recovery without deformity.

FIG. 4. Patient was a fifteen year old boy. X-ray film shows an example of complete separation of the proximal humeral epiphysis without any triangular fragment remaining attached to the epiphysis. The patient was treated using sling and swathe for eleven days; however, 3 cm. of shortening developed although full function was regained.

textbook state that their routine treatment is reduction by manipulation followed with application of a hanging cast. As alternate methods of treatment they suggest manipulative reduction followed with immobilization with the arm elevated 60 to 90 degrees and either in forward flexion or external rotation. On the same page they state that "If satisfactory reduction cannot be accomplished and if the fragments are not impacted, the lesion is best treated with recumbency and lateral traction." Also on the same page they say that if impaction cannot be broken up, if it is impossible to secure approximate anatomic reduction or if the fragments have united with deformity, open reduction is indicated. They also state that they are inclined to perform open reduction in children for this lesion more frequently than for fractures in the same region in adults because they believe "It is important that anatomic reduction be secured." After open reduction they immobilize

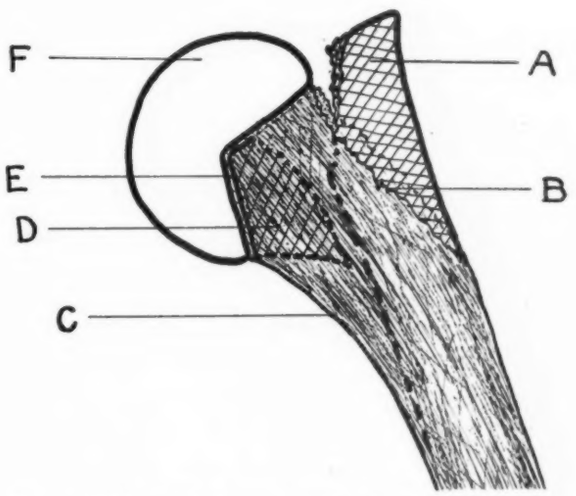


FIG. 5. This semi-diagrammatic sketch of the upper humerus showing a fracture separation of the proximal epiphysis reveals: A, the denuded upper end of the shaft which has become extruded through a tear in the periosteum at (B); C, the periosteum on the inner side, stripped away from the shaft; D, the triangular fragment of diaphysis remaining attached to the epiphysis; E, the epiphyseal plate; F, the articular head of the humerus. Compare this sketch with Figure 8A, C, D and E.

the arm in a Velpeau dressing or with Kirschner wires passed through the skin, humeral head and into the shaft.

At the other extreme is Blount,[3] who states, "The greatest fallacy is to think that accurate reduction of an epiphyseal fracture at the proximal end of the humerus is important enough to require open reduction." He says this can be reduced usually (if seen early) by traction and hanging cast, but occasionally it is difficult to reduce it and maintain reduction unless the arm is placed over the head in the pivotal position and fixed there in a light plaster spica for four to five weeks.

Stewart and Hundley[13] mention the injury to say that the adduction type is the most common seen in the upper humerus in children, that it must be reduced accurately and that the biceps tendon is often interposed between the fragments. The authors believe open reduction is necessary if manipulation fails. They do not state how much displacement makes manipulation necessary or how accurate open reduction and internal fixation must be. Their one illustration with a five-week follow-up shows far from perfect realignment and not enough elapsed time to show if this might have been corrected by growth or developed a growth disturbance.

ANALYSIS

Our forty-three cases have been divided into four groups according to the degree of displacement seen on x-ray films, i.e., 1 plus to 4 plus. This classification must, of course, be rather arbitrary due to inability to catch the humerus always in the same amount of rotation which in itself may alter the apparent displacement. The study therefore can be divided into sixteen cases with 1 plus displacement, ten with 2 plus, six with 3 plus, and eleven with 4 plus displacement. The sex and age incidence is as follows: males to females 3:1; males averaged thirteen years of age and females averaged eleven years of age. The right side was involved seventeen times, the left side twenty-six times. The etiology consisted of three main groups: (1) falls (in general), (2) sports (football, wrestling, etc.), and (3) traffic accidents. Twenty-eight patients stated definitely on admission that they had fallen *on* the arm or shoulder or had been hit there; this cause differs from the usual given textbook etiology of a fall on the outstretched hand. The height of the falls (where stated) ranged from 10 to 15 feet, and four other patients fell from trees of unstated height. As for age incidence, our youngest patient was five years old and the oldest was seventeen years of age. Eight patients were in the age group of five to nine years, whereas thirty-five patients were in the age group of ten to seventeen years.

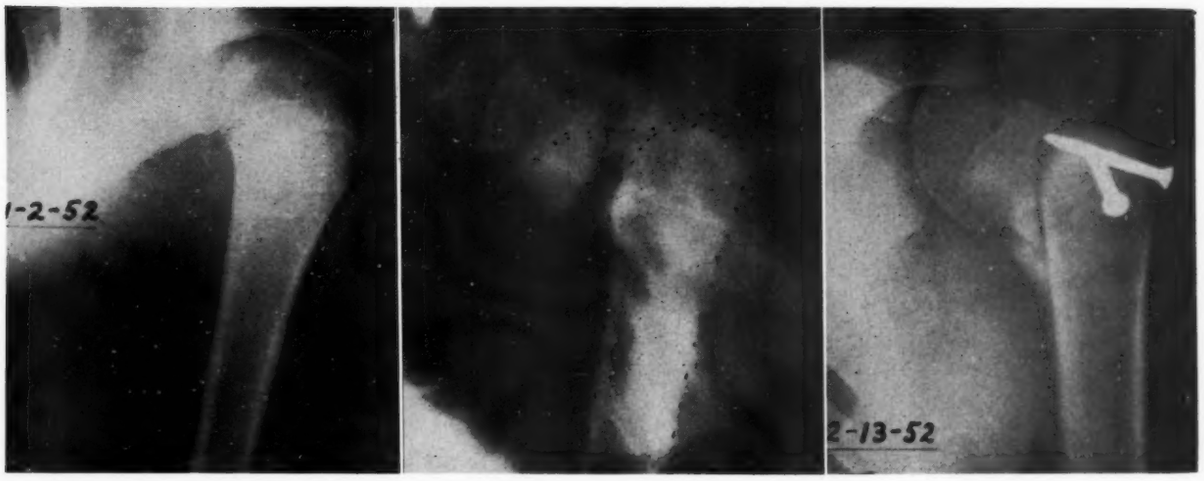
The injury therefore occurs chiefly in older children who are prone to undertake the rougher types of sports or who in general fall from greater heights than those sustaining one of the more common wrist or elbow fractures.

ASSOCIATED LESIONS

Associated lesions are uncommon and in this group there were only three in which there was any significance from the standpoint of treatment or eventual outcome. One patient, J. G., a six year old boy, sustained an injury in traffic. This boy had a fracture of the shaft of the humerus which was plated, but the epiphysis with 2 plus displacement was treated with closed reduction and plaster; he also had a fracture of the jaw and paralysis of the radial nerve. This boy remained in the hospital two and a half months because of his associated lesions. The second case (P. F., a ten year old boy), was an old "polio" case with flail shoulder joint previously fused for stabilization and with permanent shortening; a spica was used for two months to get bony union and restiffening of the shoulder. The third patient (J. F., a twelve year old boy) had an extensive, severe poison ivy rash covering his entire shoulder; this rash precluded open reduction. (See case history of this patient.)

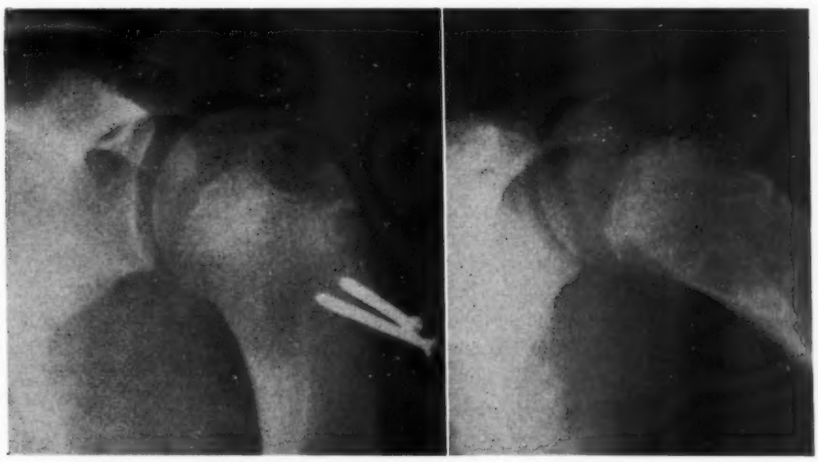
TREATMENT

Twenty-four patients were treated with ambulatory measures, ten additional with hospital admission and conservative measures, and nine with hospital admission, conservative measures and open reduction. Conservative treatment consisted of attempted reduction under anesthesia in twenty-two patients; traction and suspension by means of Kirschner wire with the arm held over the body in fourteen patients; and plaster splints or casts in sixteen patients; or by sling and swathe only. Exercises in the non-operative cases were



6A 6B 6C

FIG. 6A, B and C. Patient was a fourteen year old boy. A, the original anteroposterior x-ray taken on the day of injury. There does not appear to be any deformity in this view. The fracture was missed clinically as well as by x-ray and the boy was sent home with his arm in a sling. He returned the following day. 3, an x-ray was taken in the transthoracic view showing the position of the humeral head and the shaft, and marked displacement at the epiphyseal line. He was operated upon the same day; open reduction was performed and the shaft was fastened to the triangular fragment with two screws. The screws were too short and fixation was lost. C, an anteroposterior view in external rotation taken five weeks after the original operation shows the fracture healing with recurrent deformity following loss of fixation.



6D 6E

FIG. 6D and E. Same patient. D, a similar view, shows the same humerus seventeen months after the original injury. Note the correction of the deformity. Also note that the screws are projecting from the shaft. It would seem at first that these screws had worked themselves loose; however, on studying the x-ray films more carefully it is seen that the original upper end of the shaft which had been displaced, and in which the screws were placed, has become absorbed beneath the heads of the screws and the space between the periosteum and shaft has filled in with new bone. These screws became prominent beneath the deltoid, caused irritation and formed an adventitious bursa, giving discomfort and crepitation on motion. They were therefore removed. E, the same humerus two weeks after removal of the screws. This patient has full return of function and no visible deformity but there is a shortening of 1.5 cm. in the humerus.

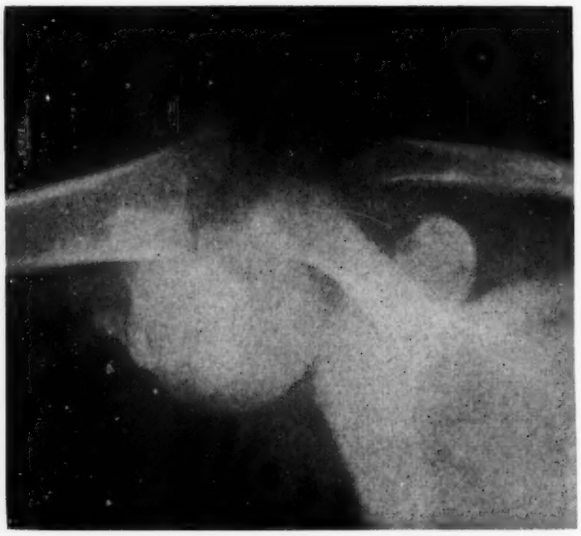


FIG. 7A. Patient was an eight year old boy. Anteroposterior view of the original fracture with marked comminution of the upper shaft and diaphyseal fragment attached to the epiphysis. This boy was treated with manipulation, traction and suspension; subsequently a sling was used. No better position could be obtained from the x-ray standpoint.

FIG. 7B. Same patient. X-ray shows the same humerus twenty-two months after the original injury. Note the marked correction of the original deformity.

started at 12 plus days on the average, and at 19 days in the operative cases.

OPERATION

Open reduction was performed in nine patients mainly because attempted closed reduction was unsatisfactory in eight of these. The outstanding pathologic conditions noted at operation were: (1) marked soft tissue damage and periosteal stripping; (2) "buttonholing" of the shaft through periosteum necessitating splitting of the latter to obtain reduction; (3) biceps tendon caught between fragments in three cases; and (4) portions of deltoid and subscapularis muscle interposed between fragments. In three cases no pathologic condition was described.

Internal fixation (in the nine operative cases) consisted of the use of one to three screws in six cases (and in one, an additional wire loop). In attempting to obtain internal fixation with screws, these were passed from the upper end of the shaft medially into the triangular diaphyseal fragment attached to the epiphysis. No attempt was made in any case to pass the screw across or through the epiphyseal plate. Chromic sutures were used in one case. In two cases no internal fixation was employed except suture of periosteum after open reduction. Two patients were operated upon as early as one day after injury, the other patients mostly at three to five days, and one as late as twelve days after injury. Four patients were kept in Kirschner wire traction-suspension after operation; three were put in plaster and two were given a sling and body swathe.

Loss of fixation or reduction occurred in four of the nine patients operated upon; two of these four had no internal fixation employed, and two had internal screw fixation (the screws being too short). (Fig. 6A to E.) Four patients subsequently had secondary operations for removal of the screws.

RESULTS

The results in general have all been excellent from the standpoint of function and use. As for the anatomic result, two main deformities have been noted, namely, (1) slight angulation or prominence of the upper humeral shaft and (2) measurable shortening. Although actual measurements were not recorded in all on follow-up visits, ten patients of the forty-three had definite shortening. Two of these patients had had operation and the shortening was 7.5 and 1.5 cm., respectively; the remaining eight patients showed shortening varying from 1.0 to 3.0 cm. Therefore, some shortening resulted in at least 20 per cent of all the pa-

tients. The patient with the greatest shortening was the youngest patient operated upon (age seven years).

Of the severely displaced 4 plus fractures not operated upon, two were in a five and a half and an eight year old child, both with marked comminution (7A and B); a third was in the previously mentioned twelve year old boy with the poison ivy rash which prevented operation. This latter patient is so interesting from the standpoint of his history, lack of reduction, correction of his early deformity and recovery of function, that I believe his case history should be included.

J. F., a twelve year old schoolboy, fell 15 feet from a tree on September 2, 1943. This accident resulted in immediate deformity, pain, swelling and disability in his left arm and shoulder. In another city two forceful attempts at closed manipulative reduction were made after x-ray examination on the day of injury and three days later, both without success. The parents were informed that a tendon lying between the fragments prevented reduction and that an open reduction must be performed. He was therefore referred to us on September 6th and was admitted to the Presbyterian Hospital.

When the boy was examined, the left arm and shoulder were supported in a sling and body swathe. The left humerus was about 2.5 cm. short and the upper end of the shaft was displaced forward and upward. There was marked swelling and ecchymosis over the entire shoulder region, upper arm and chest. Any attempt at motion caused severe pain. The skin of this entire area was covered with a rash, bullae and scratch marks. This boy had had a rather extensive poison ivy rash in this region before he was injured. The radial pulse was good and the axillary, radial, median and ulnar nerves showed no impairment in function or sensation. The patient had no other symptoms or signs pertinent to his injury.

X-ray examination on September 7th showed a fracture-separation of the proximal humeral epiphysis with marked displacement of the fragments. (Fig. 8A.)

However desirable it seemed to obtain reduction, it was obvious that open reduction (for which the boy had been referred) should *not* be undertaken in view of the extensive skin rash and the great risk of wound infection, with possible subsequent prolonged suppuration, non-union, growth disturbance or other deformity. It was therefore thought best and safest to treat this particular fracture *in this patient* by all possible conservative means and to trust in "nature" to correct the deformity. The situation and why operation almost certainly insured disaster were explained carefully to the parents, and with much reluctance they agreed to the conservative program advised.

FIG. 8A. Patient J. F., a twelve year old boy. X-ray of best anteroposterior view that could be obtained upon admission.

The patient was then put to bed and his arm was suspended at his side in 60 degrees of abduction with the humerus in neutral rotation. Low intensity heat was applied and he was encouraged to practice elbow, wrist and finger exercises. The swelling at the shoulder slowly decreased. Two weeks after admission it was possible to test rotation of the humerus with palpation and without pain, and it was found that the head of the humerus rotated with the shaft. With bony union thus becoming evident, the suspension in abduction was discontinued. The arm was placed in a sling and the patient was made ambulatory and encouraged to practice pendulum exercises every two hours. The rash had now cleared and x-ray examination revealed beginning union of the fracture. He was discharged from the hospital sixteen days after admission (September 23rd) and followed up in the office.

Four weeks after admission (September 30th) it was possible for the boy to abduct the left arm to 90 degrees without pain. Three weeks later he could actively abduct it to 160 degrees; internal rotation of the humerus was possible to 70 degrees and external rotation was 60 degrees. The upper end of the humeral shaft showed a visible and palpable prominence. At this time (October 22nd) it was possible to get an x-ray film with the humerus in some external rotation. (Fig. 8B.) By the end of three and a quarter months (December 11th) the patient was playing basketball and using the

FIG. 8B. Patient J. F. Film shows same humerus seven weeks after injury, at which time it was possible to obtain a view with the humerus in external rotation. Note the marked deformity and bony healing.

FIG. 8C. Patient J. F. Film shows the humerus three months after injury. Note beginning resorption of the prominent upper end of the shaft. (Because of incomplete external rotation, this view is not entirely comparable to the previous view.)

FIG. 8D. Patient J. F. Anteroposterior view six months after the injury. This discloses marked absorption of the upper end of the diaphysis.

FIG. 8E. Patient J. F. Same humerus thirteen months following the initial injury Note the marked correction of the deformity and the slight residual bowing of the upper humeral shaft.

horizontal bar in the school gymnasium. (Fig. 8C.) By six months after injury (March 4, 1944) he was enjoying full use of the arm and shoulder without pain. His total abduction was limited to 165 degrees. The projecting end of the upper shaft was no longer prominent. (Fig. 8D.) One year following injury (September 27, 1944) the only clinical deformity to be detected was slight anterior convex bowing of the upper humeral shaft. (Fig. 8E.) This patient subsequently played college football and tennis, and enjoyed swimming without any discomfort or handicap whatsoever. When the patient was last examined eleven years and three months after injury (December 4, 1954), a 2 cm. shortening of the left humerus was found by measurement, but there was no loss in strength, motion range or function and he carried on full physical activity without complaint or pain.

CONCLUSIONS

1. The results (functional and anatomic) have been so excellent in the mild to moderately displaced cases (twenty-six) that the recommended treatment need be no more than rest in a sling and swathe for two weeks, followed with early active exercises and use. Two patients only in this group showed shortening of more than 1.0 cm. These were the two patients whose epiphyses were displaced from an intact diaphysis without a triangular fragment remaining attached.

2. The results in the moderate to severely displaced cases (seventeen) were excellent from the standpoint of function. Of this seventeen, five showed shortening (one of 7.5 cm. in a seven year old girl who underwent surgery). Three others showed mild anterior prominence or bowing of the upper humeral shaft.

3. As for operative treatment, it would seem from our findings that little or nothing has been accomplished when we compare reductions or those patients successfully operated upon with those receiving no reduction or those operated upon and in whom loss of reduction and internal fixation resulted.

One might well pose the question: In patients under fifteen years of age with marked displacement should we keep them comfortable and let "nature" correct the deformity or should we try to find a more satisfactory type of internal fixation such as two or three Kirschner wire implants placed at different angles? This might be reserved for the older patients (twelve to seventeen years of age) in whom attempted closed reduction or traction and suspension has failed and in whom interposition of the biceps tendon is strongly suspected.

It might perhaps be better to treat the markedly displaced fractures simply by attempted closed reduction and follow this by suspension of the arm in 70 to 80 degrees of abduction for two weeks. Early pendulum exercises would be started after this.

From our study of these patients, their x-ray films and their individual long-term follow-up observations, several points become very obvious and should be emphasized: (1) The injury appears on its face far more serious than it actually is. (2) Bony healing occurs rapidly. (3) Essentially full recovery of shoulder and arm function results. (4) By subsequent growth the greater part of the original angular deformity corrects itself. This fortunately is true regardless of the fracture being left uncorrected, or after loss of correction following closed or open reduction. Shortening occurs in approximately 20 per cent of these humeri regardless of their treatment.

SUMMARY

In our hands the foregoing examples demonstrate all too clearly how overconscientious we have been in handling this "much overtreated fracture." In the future I hope we may be able to adopt a routine course of treatment bordering on a laissez-faire policy, and I suspect the next generation of fracture surgeons will find the late follow-up results quite as good or better than those in our study.

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Colles' Fracture—Residual Disability

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COLLES' fracture, contrary to popular belief, is still a difficult fracture that results in great residual disability. The fracture too often is considered lightly and is, therefore, inadequately treated, leaving the patient with limitation of motion and a weak, disabled, deformed and painful hand and wrist. It seems commonplace to accept less than the best obtainable result in the treatment of this fracture, particularly in the older age group.

Bacorn and Kurtzke reported a series of 2,132 cases of Colles' fracture recorded by the New York Compensation Board, in which only 2.9 per cent were judged to have no permanent disability and, "The average disability regardless of the age, adequacy of treatment or severity of fracture was 24 per cent permanent loss of function of the hand." This appeared to be a very great residual disability. There are many other series in the literature which report much more desirable results. In some there is almost complete reversal of Bacorn and Kurtzke's result, with up to 95 per cent good functional results. Gartland and Werley[10] very carefully analyzed a series of sixty cases which were studied an average of eighteen months after fracture, and found that 68.3 per cent were satisfactory and 31.7 per cent were unsatisfactory. DePalma[8] reported excellent results in a series which were treated with internal fixation.

The wide variation in reported results prompted us to investigate our series and to consider carefully the residual loss of function as determined by standards of disability evaluation. The results in our private series reveal that residual disability is higher than previously thought. Again, reviewing several of the reported series, it is suspected that if a more rigid standard of grading had been used, the results would also have been less good. Certainly many patients in those groups classified as good and satisfactory would have had moderate disability. In discussing the matter with representatives of four insurance companies carrying compensation coverage, it was learned that in each case of Colles' fracture a reserve is established to pay for an expected residual disability.

It is the purpose of this paper to report a series of seventy-five cases treated in private practice and to make suggestions for the reduction of residual disability. A further series of fifty-nine cases which we did not treat but which were recorded by the South Carolina Industrial Commission were studied. The disability rating in this group, in which patients were treated by many physicians throughout the State, was unusually low. The available data in these cases were insufficient to make this study of any value.

RESULTS

In the series of private cases there were seventy-five Colles' fractures, with a usual follow-up of twelve to sixteen weeks. Colles' fracture, as included in this group, depicts a fracture of the distal end of the radius with dorsal angulation and lateral deviation of the distal fragment. In ten cases (13.3 per cent) there were excellent end results from both x-ray and clinical standpoints. The range of motion was normal and the final x-ray appearance was that of a normal wrist. There was no residual dorsal angulation or radial deviation, no shortening and no pain. (Fig. 1.)

Fifty-two patients (69.3 per cent) had from 5 to 20 degrees residual variation from the normal volar angulation of 10 degrees. Almost without variation those who had a residual dorsal tilt had residual radial deviation and some shortening. The cosmetic appearance of the deformity was noticeable only to the trained eye. The range of motion was good. The remaining discomfort was mild. In spite of the final x-ray and clinical changes found in this group, the functional results were considered satisfactory. According to the standards of disability evaluation using McBride's plan, it was calculated that this group had an average of 14 per cent permanent loss of function, the disability varying from 5 per cent to 25 per cent. (Figs. 2 and 3.)

1A 1B 1C

FIG. 1. A, x-ray showing simple Colles' fracture in a forty-eight year old white woman. B, reduction film shows 10 degree volar angulation and 22 degree medial inclination of radius. C, eleven weeks later, film shows 10 degree volar angulation and 22 degree medial inclination of radius. No shortening, limitation of motion, pain, deformity or residual disability was present.

2A 2B 2C

FIG. 2. A, x-ray showing simple Colles' fracture in a forty-seven year old white woman. B, reduction film shows 5 degree volar angulation and 20 degree medial inclination of radius. C, four years later, film shows 5 degree dorsal angulation and 18 degree medial inclination of radius. Loss of motion: 20 degree volar flexion and 10 degree ulnar flexion. Pain on twisting motion of wrist was present with no obvious deformity; 10 per cent permanent loss of function of hand.

3A 3B 3C

FIG. 3. A, x-ray showing comminuted Colles' fracture extending into the wrist joint of a fifty-seven year old woman. B, reduction film shows 10 degree volar angulation and 20 degree medial inclination of radius. C, ten months later film shows 10 degree volar angulation and 20 degree medial inclination of radius. Non-union of ulnar styloid; 4 mm. shortening of radius was noted. Limitation of motion: 15 degree volar flexion, 15 degree dorsal flexion, 5 degree radial and ulnar flexion and 10 degree supination. Moderate pain and discomfort were present; 20 per cent permanent loss of function of hand.

4A 4B

4C 4D

FIG. 4. Poor results in a case of Colles' fracture. All patients had more than 30 per cent permanent loss of function of hand. A, 11 degree dorsal tilt, 13 degree medial inclination of radius and 5 mm. shortening. Pain in wrist and limited motion were present. B, moderate osteoporosis of bone. Mild loss of volar tilt and 3 mm. shortening. Pain and limited wrist motion were present. C, fracture through old poorly reduced Colles' fracture. D, poor reduction with considerable shortening.

Thirteen cases (17.3 per cent) had more than 20 degree variation from the normal volar tilt, and associated with this were varying degrees of radial deviation and shortening. This group was considered unsatisfactory. There was mild to moderate residual deformity, some limitation of wrist motion and some residual discomfort. Three patients had stiffness of the fingers. One patient had division of the extensor pollicis longus tendon occurring three weeks after injury. It was calculated that this group had an average of 34 per cent permanent

5A 5B 5C

FIG. 5. A, x-ray showing comminuted Colles' fracture in a sixty-five year old white woman. B, reduction film shows 25 degree volar angulation and 20 degree medial inclination of radius. C, twenty-two months later, film shows 20 degree volar angulation and 20 degree medial inclination of radius. End result was full motion, no deformity, no pain and no permanent loss of function.

disability, with variation from 25 to 50 per cent. (Fig. 4.) The entire group of 75 fractures had a 17.7 per cent residual disability.

Females in the series were in great preponderance (89 per cent). The age groups followed the usual pattern seen in private practice, with 79 per cent above forty years and 56 per cent above fifty years. One-third of the fractures were non-comminuted, two-thirds were comminuted, and in half of these the fracture extended into the wrist joint. The end result in the younger age group and in those patients with non-comminuted fractures was generally better.

COMMENTS

Reduction of the fracture remains the most important single factor in obtaining good results. Colles, who described the fracture in 1814, stated, "If a fracture of the lower end of the radius be badly set, the patient will not get the use of his fingers for a long time." In order to get good reduction adequate anesthesia is necessary. Proper reduction can never be obtained by "snapping it back" without an anesthetic. Gentle but firm manipulation is necessary. The soft tissues, including the nerves and vessels, have to be carefully protected from further damage. Rocking of the distal fragment tends to comminute the fracture further and to damage soft tissue. Traction in the long axis of the radius should be applied, after which the distal fragment is manipulated into place. The pronated position of the forearm, 20 degrees of volar flexion of the wrist, and ulnar deviation of a comfortable tension are the other factors considered in the reduction. The use of mechanical aids may serve only to complicate reduction. In spite of perfect or near perfect reduction there is a great tendency for angulation and shortening of the distal fragment. In our series nine fractures were overcorrected in volar angulation an average of 10 degrees. Five of these were considered excellent results and four were termed good results. We observed that there was less tendency for loss of position in those fractures which were overcorrected. (Figs. 5, 6 and 7.) A portable postreduction x-ray should be made while the patient is under anesthesia. Remanipulation should be carried out immediately if necessary. In the presented series fourteen remanipulations were done.

Immobilization of Colles' fracture is far from standardized and there is probably no single type which will hold all reduced fractures in good position until healing occurs. Short arm dorsal and volar splints, short circular plaster, and circular plaster extending to above the elbow all are used with varying degrees of success. Even in some of the reported cases in which the distal radial fragment was fixed to the ulna by a threaded wire there was some loss of position during the healing period. In our group the sugar tong splint, held in place by an elastic bandage, was used. It was thought that reduction forces could best be maintained during the application of this splint than with any other type of fixation. Additional advantages of the sugar tong splint are easier control of swelling, maintenance of the

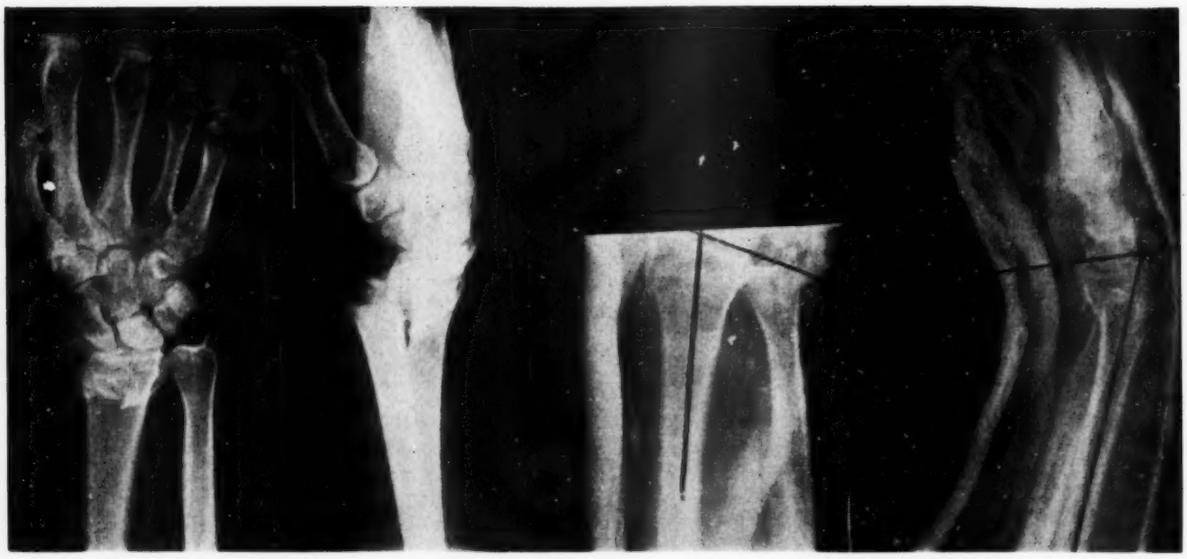


FIG. 6. Thirty-nine year old white woman with a comminuted Colles' fracture. Reduction film shows 20 degree volar angulation.

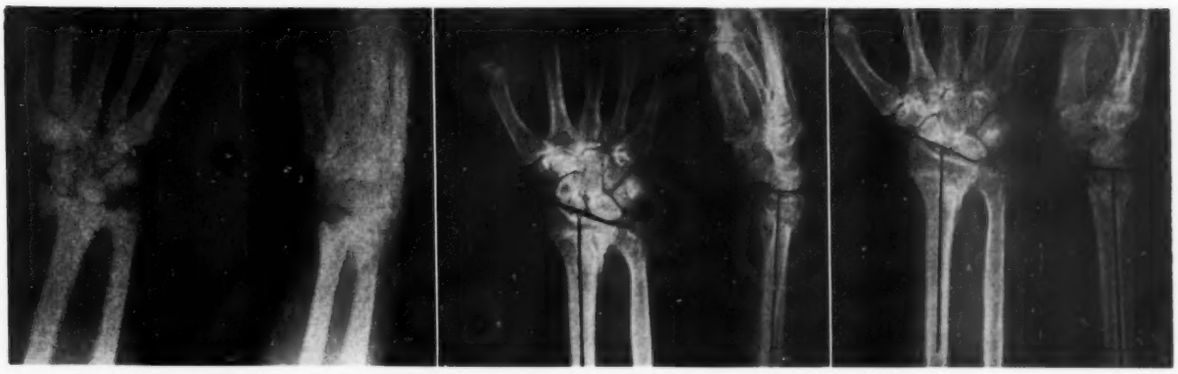
7A 7B 7C

FIG. 7. A, same patient as in Figure 6, seven days later; film shows volar angulation of 23 degrees. B, remanipulation with resultant volar angulation of 4 degrees. C, film taken seven weeks later showing 12 degree dorsal angulation.

pronated position, more rigid fixation than the short arm cast or splint and retention of some elbow motion. The sugar tong splint extends from the palmar crease, around the elbow, to the metacarpophalangeal joint on the dorsum of the hand. Careful molding of the splint between the thumb and index finger maintains ulnar deviation by pressure over the second metacarpal.

The period of immobilization should be continued until the fragments are firmly adherent and no further absorption will take place at the fracture site. This usually requires from six to eight weeks. Increased angulation and shortening can occur even after six weeks' immobilization. (Fig. 8.) If there is considerable tenderness at the fracture site after removal of the splint, some type of immobilization should be reapplied. It is probable that the increased period of immobilization will not materially alter the return of wrist motion. In our series the period of immobilization averaged six and a half weeks.

The control of swelling is too seldom stressed. Edematous fluid, if allowed to remain in any tissue long enough, will result in fibrosis of that



8A 8B 8C

FIG. 8. A, x-ray shows comminuted Colles' fracture in a sixty-five year old white woman. B, 3 degree volar angulation when sugar tong splint was removed seven weeks after injury. C, five degree dorsal angulation (loss of 8 degrees) seen seven weeks later.

part. The small joints of the hand are particularly vulnerable to this process, and only a few days are required for the process to become irreversible. Those joints already damaged by arthritic processes are particularly vulnerable. Once the process of organization of edematous fluid is established, it is not inconceivable that a frozen hand will be the end result. It is noted in Bacorn and Kurtzke's report of the 2,000 cases of Colles' fracture that the average patient with 25 per cent disability had, in addition to other factors, mild flexion defect of the distal interphalangeal joint of the index, middle and ring fingers. The most likely cause for this limitation in the Colles' fracture is the arthrofibrotic process secondary to prolonged swelling. In using the sugar tong splint, release of constricting forces is easily controlled by loosening the bandage and then tightening it as swelling subsides. Pressure over the dorsum of the hand constricting the lymphatics should be carefully avoided.

Roentgenograms, in addition to those taken before and immediately after reduction, should be made when swelling subsides. Further x-ray study should be done approximately two weeks following reduction. If there is any loss of position, it can and should be corrected when discovered.

Physical therapy is instituted as soon as the patient awakens from the anesthetic, and this therapy is continued throughout the course of treatment in the form of full finger and shoulder motion and limited elbow motion. Professional physiotherapy is rarely needed.

CONCLUSIONS

1. Residual disability from Colles' fracture is greater than generally realized when it is rated on the basis of accepted disability evaluation standards.
2. Perfect reduction and maintenance of this reduction during healing is still the prime essential in obtaining excellent results.
3. Frequent x-ray studies are necessary to determine maintenance of position of the fragments.
4. Mild overcorrection is more desirable than minimal undercorrection.
5. Prevention of swelling of the small joints of the hand has been insufficiently stressed. The resultant arthrofibrosis is often more crippling than the fracture.
6. The sugar tong splint has a place in the treatment of Colles' fracture.
7. Early finger and shoulder motion is again stressed.
8. The period of immobilization should be individualized on the basis of clinical and x-ray findings.
9. Each Colles' fracture should be treated as if a final evaluation of disability were required.

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DISCUSSION OF PAPERS BY DRS. SCHENK, SMITH AND STEPHENS, JEWETT, SMITH, AND GREEN AND GAY

Roswell K. Brown (Buffalo, N. Y.): The paper on fractures of the hip by Drs. Schenk, Smith and Stephens presents interesting statistics which, as they say, are not statistically significant. However, there is one important statistic which is hard to accept; we are still losing one-third of the patients whom we treat for fractures of the hip.

I want to mention three points. The emergency treatment of fractures of the hip was abandoned early because of poor fixation and because of the high mortality rate. Was this conclusion statistically sound or were we just frightened by a few early poor results without enough data to make sure whether the method was better or not?

We should commend these workers for directing attention not to position, union or anything in these fractures except the mortality. Survival is the important problem. When an older person breaks her hip, the question is not whether the hip will be deformed or if the hip will unite, but rather, if the patient is going to die.

Perhaps the inconclusive nature of existing data is due to the fact that so many unrelated factors are involved. Many of us were impressed with the story of Dr. Gatewood's second case. Perhaps this patient was in the process of an illness which later became fatal, and perhaps she fell and broke her hip because of this other illness. This thought was brought out quite clearly by several discussers: by Dr. Bisgard, Dr. Wagner, Dr. Wade and Dr. Gatewood himself.

Dr. Jewett mentioned the fact that persons have strokes and other illnesses which cause them to have fractures. I would like to ask Drs. Schenk, Smith and Stephens if they can devise any way of separating hip fracture cases into two groups, namely: (1) those in which the patient is seriously ill because of an accidental hip fracture, and (2) those in which the patient has a hip fracture as one of the incidents in the process of dying.

My last question is: Do these relatively indefinite data mean that we would be warranted in making a controlled series of these patients in which alternate cases would be operated upon and the rest treated with conservative, supportive measures?

Walter D. Ludlum (New York, N. Y.): I would like to address a few remarks particularly to the discussion of the papers on fractures of the hip.

In the first paper, a series of 500 cases, in which hip fractures apparently intra- and extracapsular are grouped together, there is a rather striking similarity over the ten-year period of the mortality emphasized. The ultimate outcome was evaluated in terms of mortality rather than emphasizing the end results from a functional standpoint.

It seems to me that the mortality, the 33⅓ per cent or approximately one-third, is higher than that of which most of us are aware. To group intra- and extracapsular fractures in this particular way is a difficult concept for me. Perhaps, as one author has already done, such cases should be studied as to operated and non-operated cases.

Few of us now would consider, I believe, not operating upon a patient with an intracapsular fracture. Perhaps we would for an extracapsular fracture. However, the intracapsular fracture, if the case warrants any treatment at all other than pure neglect of the fracture itself, does warrant the relatively simple technics of reduction and internal fixation.

We will admit that if there is no operation for the extracapsular fracture there will be good functional results and perhaps a fair percentage of survival. But with an intracapsular fracture, unless it is impacted, the surgeon might as well accept a bad result if he does not operate. Therefore I cannot follow the aforementioned concept.

Finally, regarding the mortality rate of one-third, we are conscious that appraisal of mortality by different authors is differently made. I quite go

along with Schenck and his co-workers in their use of hospital mortality rates as a guide for evaluation of cases.

At least the surgeon has a full knowledge of his patients while they are in the hospital, and whether they average three or eight weeks in the hospital at least a fair appraisal of the mortality can be given. However, in our hospital, where we follow rather closely both the intracapsular and extracapsular fractures, hospital mortality from whatever cause, whether it is completely unrelated to the fracture or not, is decidedly less than one-third. I think it is so reported in many institutions.

I followed with great interest, also, the details of Dr. Jewett's paper and the excellent results that he is getting with union, above 85 per cent. In our group we are not regularly accustomed to supporting the nail with some extension along the shaft, and we have seen a number of cases in which the proximal end of the nail projects into the hip joint. I wonder, then, if the concept of fixation along the shaft is a fully satisfactory one or whether we endanger the articulation itself too much by too firmly supporting the distal end of the nail.

Peter B. Wright (Orlando, Fla.): Dr. Schenk's statistics were very interesting; however, I think, too, that the 33⅓ per cent mortality rate is astounding.

I am just wondering what is meant by conservative treatment of the hip. It seems to me that the conservative method is early fixation. I think that unless the individual patients are extremists there are no contraindications to giving them the benefit of firm fixation and early ambulation or sitting and turning.

Dr. Jewett stressed a good point when he said that he required good reduction and firm fixation, and also that his patients are individualized and each case is considered a problem within itself.

In epiphyseal separations of the proximal end of the humerus I think the important thing is to be aware of the likelihood of growth discrepancy and the necessity of telling the parents to expect it.

I was glad to see Dr. Green refer back to the old masters. If we will read the original articles by some of these men, we will find that meticulous treatment and meticulous postoperative care was their real motto.

Joseph DeSault, speaking of supracondylar fractures of the humerus, stated that manipulation, complete reduction, tightening the splint, loosening it for swelling and tightening it again as the swelling subsides, requires a lot of time; yet by doing so we will accomplish much better results.

Harrison Lloyd McLaughlin (New York, N. Y.): An in-hospital mortality rate of 33⅓ per cent for patients with fractures of the hip is somewhat surprising in that the average seems to range between 5 and 10 per cent for fractures of the neck of the femur, and from 15 to 20 per cent for intertrochanteric fractures. Mortality is not the direct result of hip fracture but is generally caused by stasis of a depleted geriatric physiologic condition resulting from immobilization of the bedfast elderly person by pain or, as was formerly the case, by a plaster spica or traction apparatus. Operative fixation is not necessary for the treatment of the fractured hip, *per se*, but is the only available method for stabilization of the fracture coincident with continued mobilization of the patient and maintenance of this over-all physiologic condition at a reasonably normal level throughout the healing period. This should be done as soon as possible, even at the inconvenience of deferring elective cases already on the operating schedule. Analysis of two large series of hip fractures internally fixed at our clinic demonstrated the incidence of complications to be doubled, and the morbidity and mortality to be proportionately increased with fixation carried out later than twenty-four hours following injury, as compared to those receiving fixation in the first twenty-four hours after injury.

Preston A. Wade (New York, N. Y.): I will confine my remarks to the paper by Dr. Smith on the injuries to the upper humeral epiphysis. I had the opportunity to read and study his paper some time ago and enjoyed it very much. I believe this is a real contribution, as all of Dr. Smith's papers are, to our knowledge.

The question of injury to the upper humeral epiphysis is certainly confused in the textbooks and in the minds of many surgeons who treat children's fractures. I believe this is going to be one way to document the results so that we can have some encouragement in facing a displaced fracture and allowing it to remain displaced without going to extremes to reduce it.

We all see epiphyseal injuries that have been overtreated and many that have been operated upon with sad results. The only thing about this series of cases is that it seems Dr. Smith's investigation shows that even if operated upon, these patients do have a good result; all that they may suffer is an unnecessary operative procedure and scars, and perhaps the suffering of the parents while the patients go through the various operative procedures.

It is very difficult to teach a young resident or a young man who is starting to treat fractures that children's fractures are different and that the same principles do not apply to children as to adults. The man's courage is shaken somewhat by hysteria of the family, the roentgenologist and the pediatrician who see criss-cross fractures and get terribly upset if something is not done. I think Dr. Smith's paper is very valuable in that regard.

I note that Dr. Smith did not advocate the hanging cast in the treatment of these fractures.

It would seem to me that although this is a very popular method of treatment for all injuries to the humerus there are few cases in which a hanging cast is useful. Certainly this case is not one of those. The displacement is usually the result of the fracturing force; and if the arm in hanging by the side does not replace itself without a hanging cast, it will not replace itself with a hanging cast.

I have recently listened to a report of a number of cases exactly like the author's in which the hanging cast had been used and was credited with the good result.

It seems to me that just because a good result is obtained by the use of a hanging cast it does not mean that the cast is the cause of the good result.

WILLIAM H. CASSEBAUM (New York, N. Y.): I think Dr. Green has given us a very excellent paper, one which I think we can all read with a great deal of profit. I hesitate a little, therefore, to talk about the points of disagreement.

I do not believe the results of Colles' fractures are bad. I will concede that the anatomic results are often very poor. There just is no way to hold a comminuted oblique fracture with plaster alone. Plaster is from ½ to 2 inches away from the parts it is trying to immobilize, and separated by a moving mass of muscle. Although there are a great many methods of immobilization and positioning of the wrist, I think that some fractures cannot be held by external appliances alone.

I think we have to realize that if we are going to treat fractures by a closed method we are going to have some poor anatomic results. On the other hand, I think the functional results are very good. By function, I mean the patients do not have pain and they do recover their strength and the movement of their fingers. That is true even in those patients who have been treated very badly and who have had very poor intermediate results say at eight and nine months, at which time they still have stiff fingers and are demoralized and weak. Eventually, however, they get well.

Certainly not many Colles' fractures come late to orthopedic institutions or any other place for late care. The number of Darrach operations done is very small, too, because despite shortening of the radius patients do get good function.

I think the report by the New York compensation group is just preposterous. They say Colles' fracture results in loss of 24 per cent function of the hand. I certainly would not like to lose 24 per cent of my hand, and I am sure that no one who has had a Colles' fracture would choose that alternative.

I think we have been too severe in evaluating the amount of motion. Who needs all this wrist motion? Heifetz, Toscanini, but you do not need it for surgery.

STEPHEN SYLVESTER HUDACK (Cleveland, Ohio): I would like to reenforce the brief but turgid remarks of Drs. Ludlum and McLaughlin by asking a few questions.

I would especially like to address my questions to Drs. Schenk and Jewett. The thread of life is so thin as we get older, and I should know; however, what excuse is there on the part of surgeons to delay for five and six days before they use fixation or replacement?

The collected statistics on the part of doctors who perform radical surgery indicate that actually the morbidity and mortality are reduced in early operations. Even in the hands of surgeons who think in terms of surgical physiology, the statistical probability of death in one month in fractures of the hip is about 18 per cent anyway.

Neither of the essayists has made mention of late arrival of cases or negligence of relatives.

Dr. Jewett says he has 85 per cent excellent results. He says nothing about whether it is a Pauwels' type 1, 2, 3 or X. This is important.

HOMER K. STRYKER (Kalamazoo, Mich.): I want to say a word about slipped humeral epiphysis. Several years ago while doing orthopedic work, I was called in by an elderly doctor who was disturbed by the appearance of a patient's x-rays. There was a slipped humeral epiphysis without very marked displacement.

I said, "This isn't going to be very bad. We won't need any specific treatment. Why don't you just keep it in the sling, and I think your results will be good."

He said, "No, I don't want to bother with it, Doctor. I used to be pretty good at this fracture work until they got these x-rays."

That, to me, embodies a lot of philosophy of fracture treatment. I think x-rays have been a boon to mankind, but they have certainly worried a lot of doctors. If we treat children with fractures that are not displaced very badly, we take the children to the operating room or the cast room in order to get them away from their parents. We then put a little sling around the wrist and around the neck and tell the child, "Just let your arm hang; do not try to raise it up." If the fractures are badly displaced, we find you can reduce most of them by traction at 90 degrees with a little pressure down on the upper humerus. When we think we have it reasonably reduced by feel, we may slip in three or four Kirschner wires and cut them flush with the skin; this will hold the reduction when you let the arm down. Do not be too disturbed about a little displacement.

About Colles' fracture, a big help to me is the application of a cast from about the mid-upper arm down to the wrist, letting it harden while local anesthesia is taking effect. This gives you something to hang on to when reducing the fracture. You then apply a couple of layers of plaster to

complete the cast. This procedure makes it easier for me to maintain the reduction.

W. G. Schenk, Jr. (closing): We certainly are not proud of our mortality figures. We are happy, at least, that we have elicited a little discussion, and we, almost without exception, are forced to agree with the criticisms leveled at us by all of the discussers.

Dr. Hudack mentioned the delay in operation. We can in no way condone it. We have to agree with him; there is no reason for this delay. We might also add a fact that was not in the paper; the average interval from injury to admission for the entire series was 2.7 days, so most of these patients had developed some of their complications, (bronchopneumonia, urinary infections, etc.) before they were admitted to the hospital. Many of them were in cardiac failure because they had been in bed two days at home before they got to the hospital. We thought these conditions required prior treatment.

In answer to Dr. Brown's questions, I am sure we do not have enough cases to know whether immediate treatment of this lesion would yield better results than delayed treatment. He is right; we were "scared off" by a few cases in which we tried this in this series. In answer to his question about running a controlled series, I do not think that even with these figures we would be interested in attempting to run an alternate case series which would mean the alternate patients would be put in hip spicas or traction for many months. We are influenced by what we see. In spite of our poor mortality figures, we want to keep operating in this type of case.

I have no way of differentiating between the people Dr. Brown mentioned who are seriously ill because of a fractured hip and those who are dying and also have a fractured hip.

Dr. Ludlum questioned our lumping together the neck fractures and the intertrochanteric fractures. We agree with him. Our mortality is appalling. We found in both series reported the mortality rate from the intertrochanteric fractures was higher than from the neck fractures.

Dr. Wright mentioned what he called the "conservative" treatment. I do not believe we used the term. Our non-operative cases I agree, in our early series, were called "conservative" by the surgeons at that time. "Conservative" treatment comprised use of hip spicas and prolonged traction.

We have no answer for Dr. McLaughlin. Our problem is: "Why didn't the application of the principles which he mentioned, that is everything aiming at early mobilization, lower our terrible mortality rates"?

Eugene L. Jewett (closing): I appreciate all of the comments the various discussers have made. Regarding our policy of waiting a day or two before operating upon these patients, most of these people have been lying at home in bed or in the hospital for a day or more before we are called in to see them. Many of these partients are taken care of at home for several days for a sprained hip and then, after the pain continues, they come into the hospital for x-rays.

As Dr. Schenk and his group mentioned before, we believe that it is much better to have a thorough medical checkup on these elderly patients before operating upon them. Many years ago I used to operate upon these patients as emergencies, but in going over the records I found that the policy of waiting a day or more means a better result and less mortality and morbidity.

Many of our patients who fall down and break a hip do so because they have had a slight cerebral vascular spasm or accident which was the cause of the fall. The hip was broken after they had fallen. All of these patients should have careful medical and cardiac checkups before they come to surgery.

Regarding Dr. Hudack's question about the Pauwels' 1, 2 and 3 type fractures, all of these in this series were of the types 2 and 3. As we all know, there is no distinct clear-cut line between the 2 and 3 groups, and many of these fractures are really a combination of Pauwels' 2 and 3. The same thing applies to the oft-mentioned "beaks." These beaks can be on the proximal or the distal fragments and, of course, are always at the inferior aspect of the fracture site. The point I brought out in the paper is that these beaks do not need osteotomy as several writers have stated. Many of these are broken off during the closed manipulation but they are not at all a cause of non-union if they are left intact. (This is generally true.)

Long nails do have to be put almost through the proximal articular border of the head fragment, and in some cases the nail penetrates the articular cartilage. No harm is done as long as the nail does not impinge on the upper two-fifths of the acetabulum roof where all of the weight-bearing is taken. When a nail penetrates through the head, it is usually removed after healing has taken place.

Dr. Ludlum brought up the fact that the quadrilateral space has always absorbed, which I believe has always been a truism in fracture work for many years. With the Smith-Petersen nail in place, when this area absorbs, the nail has to go either in or out if the fragments remain in apposition. Most of these nails come out laterally; and when they emerge from the subtrochanteric cortex, they have no stability at that point and a whiplash motion often results. This leads to undesirable mobility across the fracture site and non-union. As I stated in an article several years ago, the fixation of the

nail appliance at the subtrochanteric outer cortical region is just as vitally important as the fixation of the fragments at the fracture site.

Regarding the two cases in which I used a Smith-Petersen nail as well as one of my nails, they were unusual cases to be sure. Both of these operations were performed in small towns quite a distance from Orlando and I did not have the right length of my nail for the case. To be sure that I had better fixation across the fracture site as well as rigidity throughout the entire hip area, I simply added a Smith-Petersen nail above mine, and both patients went on to have excellent bony union. These two cases illustrate the same principle as putting in an Albee dowel graft above or proximal to a metallic internal fixation device, a method which has been employed by numerous surgeons for many years.

FREDERICK M. SMITH (closing): It is sometimes very difficult to persuade parents that when a perfect anatomic reduction is not obtained in one of these upper humeral fractures, their child will not be completely crippled.

This difficulty is especially true if some other doctor has said that the child must undergo surgery.

I do not believe one should "knock himself out" to try to get a perfect reduction especially by operation, and I believe that we should avoid operation upon these upper humeral epiphyses if possible.

Occasionally children may get infection in spite of antibiotic prophylaxis. We did not see any in our cases. If this is done the country over, there certainly will be some infections, and I believe that operations should be avoided.

JAMES T. GREEN (closing): Dr. Cassebaum stated that patients with Calles' fracture routinely get well and that only musicians or conductors need full wrist motion. We have a seventy-five year old orthopedic surgeon in our town, a very beloved individual, who eight years ago had a fracture of the distal end of the radius. Now he has considerable shortening of the radius and loss of normal angle; and if you are not careful, these people will live to haunt you.

Disinsertion of the Biceps Brachii

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DISINSERTION of the biceps brachii is an uncommon lesion when compared to ruptures of the long head. Gilcreest[7] in a comprehensive analysis of biceps tendon injuries, found an incidence of 3 per cent in a very large series of reported and personal cases. Dobbie,[1] however, demonstrated that the lesion is more common than a review of the literature would indicate. He obtained fifty unreported cases in 1941 by writing to members of the American Surgical Association, the American Orthopedic Association and the American Association for the Surgery of Trauma. In 1951 Lee[2] reported two cases and collected an additional twenty-one that had been reported after Dobbie's article. Since 1951 more cases have been reported, particularly in the foreign literature.[3–5]

Although many different methods of repair of this lesion have been advanced, all, with the exception of Michele's,[6] entail a great deal of dissection about the proximal radius when reinsertion to the bicipital tubercle is attained. In the cases Dobbie reported five per cent had complications. This and the observation that the results were good when the tendon was attached to the adjacent brachialis led Dobbie to advocate strongly that attempts at reinsertion be abandoned.

The method proposed in this paper is very simple and as free of danger to the vessels and nerves as the exposure necessary for attaching the tendon to the brachialis. It more nearly restores the normal function of the biceps in that the power of supination is regained, and this is important in the disability assessment in compensation cases.

The procedure is a modification of the method of repair proposed by Michele in 1951,[6] in which a knot or a bulbous end is fashioned on the disinserted tendon which is then secured in a keyslot hole fashioned in the bicipital tuberosity. The author has used this method many times with great satisfaction in ruptures of the long head of the biceps, tendon transfers about the hand and foot, and the iliopsoas at the hip. When applied to avulsions of the biceps, however, great difficulty was experienced due to the large size of the tendon, the relative smallness of the bicipital tubercle, and the complete loss of exposure when the elbow was flexed in order to get sufficient length to insert the bulbous end of the tendon into the hole of the keyslot. By substituting fascia lata, the operation becomes quite simple.

PROCEDURE

General anesthesia is preferred. The arm and leg are prepared and draped. A tourniquet is not used. A reversed lazy S incision is used, with the upper arm placed medially and the mid-portion paralleling the flexor crease. The median cubital vein is ligated and the bicipital fascia incised. The bicipital aponeurosis (lacertus fibrosus) is transected if it is not already ruptured, and the biceps tendon is retracted proximally taking care not to injure the antibrachial cutaneous nerve as it emerges between the biceps and brachioradialis. Dissection is

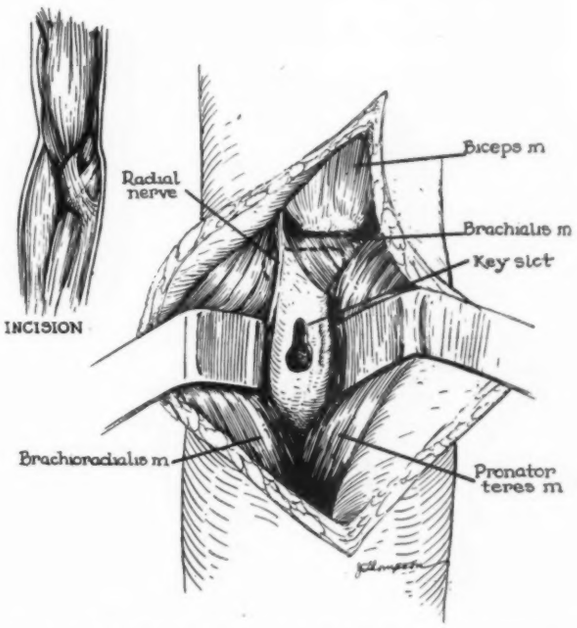

FIG. 1. The insert illustrates the incision. Exposure of the bicipital tubercle and keyslot in place. (See text.)

then carried distally between the brachioradialis and pronator teres with the forearm in full supination. The radial recurrent artery is identified, ligated and transected which allows easy medial retraction of the pronator and radial artery. The bicipital tubercle is then in

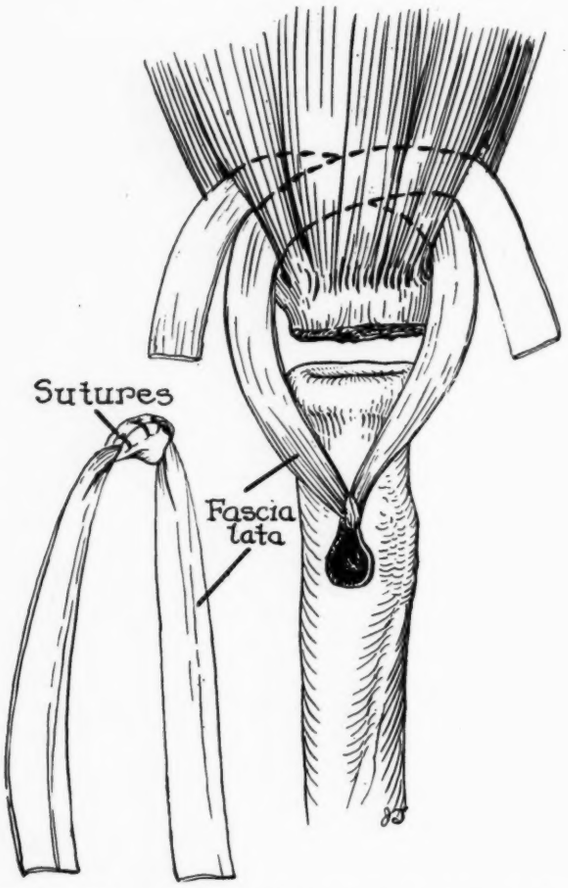

FIG. 2. Illustration of the knot in the fascial graft, its insertion into the keyslot and the ends through the biceps tendon. Actually the slot is placed in the bicipital tubercle rather than adjacent to it (incorrectly illustrated).

view and lies bare in the bottom of the wound. The radial nerve lies in a bed of fat over the capsule of the joint proximally and in the supinator radii brevis laterally. It is carried quite posterior by the supination of the forearm and is not exposed. Since retraction is restricted to the brachioradialis the nerve cannot be injured by retraction. Since there is no dissection along the neck of the radius or adjacent to the bicipital tubercle, the nerve is in no danger of being damaged. (Fig. 1.)

A strip of fascia lata 1 inch in width and 7 or 8 inches in length is obtained with the stripper. A knot is tied near its mid-point and several fine cotton sutures are used to securely hold the knot in a compact bulb. A hole is then drilled as medially and posteriorly as possible in the bicipital tubercle using a $\frac{3}{16}$ inch drill point. The hole is through the cortex only.

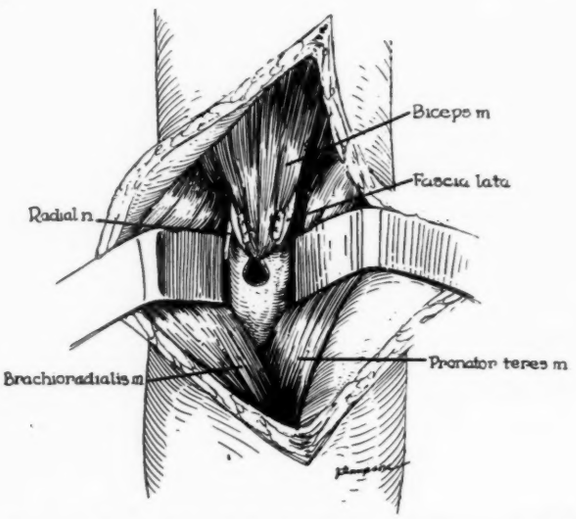

FIG. 3. Illustration of the tendon of the biceps pulled down to the slot and the ends of the graft fixed with sutures.

Another hole is drilled 5 mm. proximally using a $\frac{3}{64}$-inch drill point, and the intervening cortical bone is removed forming a keyslot. The cancellous bone under the slot is removed with a curved curet and the bulbous end of the fascia lata graft is forced into the hole portion of the keyslot and the neck pulled up into the proximal slot. Thus, the graft is securely fixed to the radius and cannot be disengaged except by pulling the graft distally to get the bulb in the hole of the keyslot. (Fig. 2.)

The retractors can then be removed. The distal end of the biceps is freshened and a transverse slit is made through the tendon about $1\frac{1}{2}$ inches proximal to the end. The two free ends of the fascia lata graft are pulled through this opening in opposite directions. The end of the biceps is pulled to the bicipital tuberosity by pulling on each end of the fascial graft. (Fig. 3.)

The tendon is secured to the fascial graft with several cotton sutures. Since these sutures are placed at points of emergence of the graft from the tendon this is $1\frac{1}{2}$ inches above the tubercle and there is no necessity for retraction or difficulty in placing the sutures. The end of

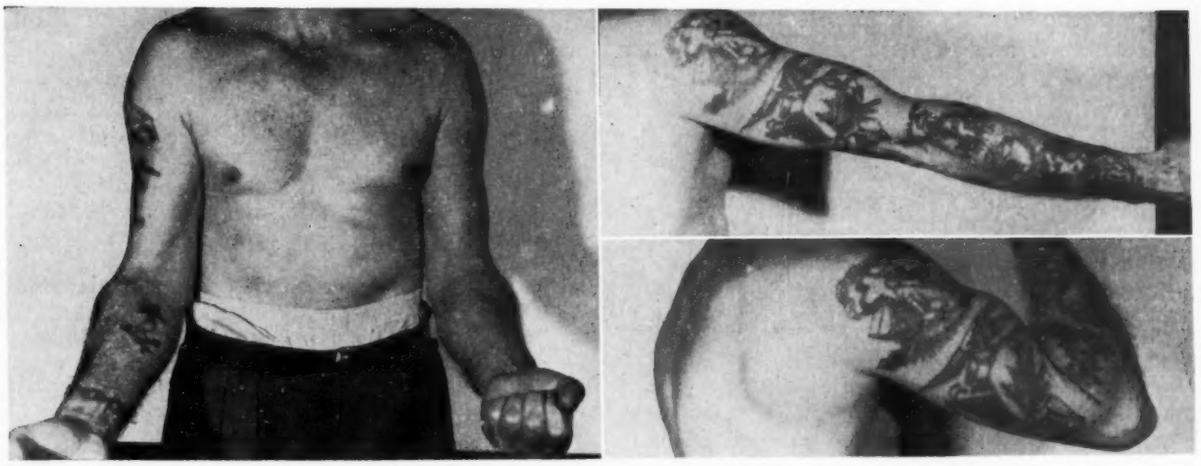

FIG. 4. Case 2. Repaired in 1951. Result four years after repair. Full motion with equal power of supination and flexion.

the tendon is clasped between the fascial graft and deep sutures are not necessary. The ends of the graft are buried in the adjacent biceps. (Fig. 3.)

The repair can then be tested safely by extending the elbow gently. The wound falls together and is closed with subcutaneous and cutaneous sutures. A plaster of paris splint is applied with the elbow at right angles and the forearm in mid-position.

Shoulder and finger motion are started immediately. After one week pronation and supination are encouraged in the splint. After three weeks flexion of the elbow is permitted, but extension is restricted until after four weeks when the splint is discarded.

Full motion is usually regained by six weeks and unlimited use of the arm is allowed after three months. Some discomfort in the antecubital fossa may be experienced on full pronation for the first few weeks but has not persisted.

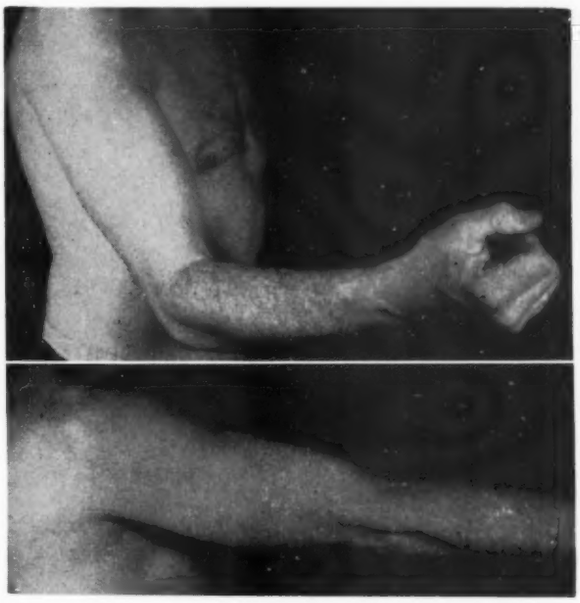

FIG. 5. Case 3. Repaired in 1951. Result four years after repair. Patient is fully employed as a shipfitter. Full motion and power of flexion and supination.

COMMENTS

The aforementioned procedure has been performed on seven consecutive patients. (Table 1.) Five have been followed; two could not be located but were followed for at least one year. The two cases illustrated were repaired in 1951. (Figs. 4 and 5.) All patients have returned to their regular occupations and have many times performed the same function with the involved arm that resulted in the original disinsertion, without recurrence.

The procedure is simple, and as one gains experience with shaping the keyslot it soon

TABLE 1

Case No.	Age (yr.)	Occupation	Date of Repair	Result
1	42	Rigger	1951	Full function, 1955
2	48	Seaman	1951	Full function, 1955
3	36	Shipfitter	1951	Full function, 1955
4	39	Seaman	1952	Full function, 1953
5	36	Seaman	1952	Full function, 1953
6	54	Welder	1954	Full function, 1955
7	37	Rigger	1954	Full function, 1955

becomes the favorite method of attaching tendons to bone. There is little danger of injury to the radial artery after release of the radial recurrent artery since no dissection about the radius is necessary. The bicipital tuberosity is usually quite bare and stands out well. All suturing is done at the wound level and the fixation of the graft to the bone is quite secure. The primary purpose of the immobilization is to limit the likelihood of ossification of any hematoma having continuity with the keyslot.

SUMMARY

1. Seven cases of avulsion or disinsertion of the distal tendon of the biceps brachii were repaired using the method of the Michele keyslot and a fascial graft.

2. The procedure is simple and yields good restoration of function.

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Operative Repair of Injuries to the Quadriceps Extensor Mechanism

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THE quadriceps extensor mechanism is an anatomic unit. The rectus femoris and the vastus lateralis, medialis and intermedius muscles coalesce into a common tendon cuff which forms the front wall of the knee joint and envelops all but the articular surface of the patella before converging to become the infrapatellar tendon. The entire mechanism acts as an extensor and stabilizer of the knee joint, and the patella is its weakest link, except in the presence of disease or excessive attritional degeneration of the adjacent tendon fibers. The most common offending trauma (violent quadriceps contraction against a sudden load of the body weight on the flexed knee) usually breaks the patella transversely across the fulcrum of the femoral condyles. The level of the fracture depends upon the degree of knee flexion at the instant of injury.

Cessation of the injuring force at that point leaves the undisplaced patellar fragments splinted by an intact tendon envelope more securely than would be possible by any surgical device. The only treatment required is for relief of pain and maintenance of function, coincident with protection against re-injury until healing occurs. But, continuance of the injuring force secondarily rips the surrounding tendon aponeurosis and quadriceps; retraction separates the fragments in proportion to the size of this tendon tear. The x-ray shows a separated fracture of the patella. The pathologic condition is a ruptured quadriceps in which part of the tear traversed the sesamoid patella.

OPERATIVE REPAIR OF FRACTURE OF THE PATELLA

It has been postulated that the patella is an unimportant vestigial remnant which may be extirpated with impunity.[1,2] This concept remains unsupported by adequate long term follow-up studies, and until this is done, it is submitted that the treatment of patellar fractures should remain predicated upon the basic assumption that this bone is a significant functional and cosmetic component of the knee joint. The objective of treatment should be to repair the bone and tendon damage in such a manner as will return the patient to his usual activities as soon and in as normal a condition as possible.

In a series of 200 fractures of the patella treated on the Fracture Service of the Presbyterian Hospital in New York seventy-two required operative repair. Two patients had bilateral fractures. Seven men and fourteen women were over sixty years of age. Forty lesions were caused by quadriceps contraction, and the remainder by direct trauma to the knee cap. The criteria for operative treatment included roentgen evidence of significant separation or comminution of the fragments, inability to maintain the knee extended against gravity, or an associated open wound. The operative technics to be discussed proved to be the simplest and most efficient methods for rapid restoration of quadriceps function in the three most serious categories of patellar fracture.

Separated transverse fractures through the body of the patella were repaired by a suture of No. 18 stainless steel wire. Sutures of a smaller caliber are prone to break with an early resumption of function. A mattress suture was passed through drill holes in the bone which, in order to prevent displacement or angulation of the fragments, should be vertical rather than horizontal. Associated lacerations of the vastus expansions were securely repaired by small interrupted silk sutures. The safety of early motion was estimated at operation by flexion of the knee prior to skin closure. If the repair were secure upon passive flexion under

anesthesia, protected function throughout the healing period proved both safe and beneficial.

Separated small fractures of the superior or inferior pole of the patella may facilitate or impede repair of the rent in the quadriceps aponeurosis. The small fragment may be excised with impunity if it is an impediment to repair, following which the tendon is sutured through drill holes to the raw surface of the body of the patella. Silk is preferred for suturing tendon to bone, and the preservation of a major fragment has proved to maintain the mechanical advantages to extension normally provided by an intact patella. Excision of the entire bone as a treatment for this type of fracture has been advocated,[3] but our results suggest this to be neither necessary nor wise.

Fractures with comminution, sufficient to preclude secure and accurate bone repair, warrant primary excision of the patella. The quadriceps aponeurosis usually is intact in continuity. Repair of the tendon defect following patellectomy is difficult to accomplish securely enough to permit early joint motion following operation, so that the disability is prolonged. Absence of the entire patella leaves the femoral condyles defenseless against direct trauma and frequently detracts, even if only to a small degree, from the efficiency and strength of the extensor mechanism. Complete patellectomy remains a procedure which should be considered as a last resort.

POSTOPERATIVE CARE

The speed and degree with which resumption of function was allowed was governed by the security of the repair as assessed at operation. Forty patients began gravity-free motion in balanced suspension within the first four days after operation. Motion in suspension was maintained for an average of twelve days. Ambulation with weight bearing, protected by splint or crutches, was begun by thirty-four patients within two weeks after operation. Unsupported ambulation was allowed for thirty patients within three weeks after operation. The speed and quality of the final results varied in direct proportion to the rapidity with which motion was resumed, and the degree to which function was maintained.

The *results* were eminently satisfactory. Fifty-nine patients have been followed for at least six months, and thirty-two patients for two years or more. Twenty-eight patients, whose fractures were repaired by suture, all returned to their former work without difficulty. Only one patient, who worked in a quarry, had any significant symptoms. The functional and anatomic results approximated normal. In patients in whom a small distal or proximal fragment had been excised, all but one had equally satisfactory results. The patient whose result was not satisfactory had an incomplete removal of a comminuted superior pole, and eventuated with residual pain requiring subsequent patellectomy.

Primary patellectomy was done twelve times. The late results have been assessed in all but one case. Ten of these patients returned to their regular work, and the eleventh was capable of doing so. Seven patients continue to complain of some weakness, instability or discomfort. Seven patients exhibited objective evidence of persistent quadriceps weakness and atrophy. Only four were free from symptoms.

QUADRICEPS REPAIR BY REMOVABLE BOLT SUTURE

Repair of major tendon ruptures by a removable wire suture was suggested in 1946, and a preliminary report submitted to this Association in 1947.[5] Since that time thirty-three major tendon ruptures, including eighteen complete disruptions of the quadriceps mechanism, have been repaired using this technic.

The *clinical material* was equally divided between the sexes, and the majority of the patients were in the sixth decade or older. The level of the rupture was proximal to the patella eight times, involved the patella five times, and traversed the infrapateller tendon five times. Eleven ruptures were repaired the day of injury, two others a few days after injury, and the other five lesions at intervals ranging from a few months to a few years following rupture. All except one have been followed from one to seven years.

The *technic*, as described in a previous report, has not been altered. Following operation the knee was progressively mobilized in balanced suspension. Once mobilized the limb was splinted in extension, usually by a circular plaster dressing. Ambulation, with weight bearing, was not only encouraged but also enforced, beginning in the most secure repairs during the second, and in less secure repairs the third or fourth week after operation. All

patients left the hospital walking and continued so throughout the healing period. The plaster dressing and the internal fixation were removed eight weeks following repair, although in several instances for no apparent reason, except possibly undue surgical caution, this period was increased. It is of interest, and undoubtedly the result of primary early mobilization and secondary continued function throughout the period of immobilization, that the range of knee motion immediately following removal of plaster and suture varied between 45 and 90 degrees.

The *results* were very satisfactory to the patients, and even more so to the surgeons, especially when assessed in comparison to other previous methods of major tendon repair. All but three patients returned to their former jobs within three to four and a half months postoperatively. One of them had never worked, and the other two were lost to follow-up. Three patients failed to regain knee flexion past a right angle, but the remainder eventuated with little or no motion defect. Only one patient complained of weakness of the limb on stairs, three complained of an occasional ache during bad weather, and the remainder were without symptoms.

Technical complications were not encountered, except in two instances in which an attempt was made to omit the use of a bolt through the tibial crest in the repair of ruptures of the infrapatellar tendon. Fixation was provided by long continuous wire loops through drill holes in the patella and tibial crest. One loop pulled out of the patella, and the other disrupted with motion.

SUMMARY

The optimum repair for a disrupted quadriceps mechanism permits continued function throughout the healing period. The results of patellectomy for fracture of the patella were inferior to those of secure repair. Excision of a small fragment of a fractured patella does not depreciate from the quality of the result, providing the tendon is snugly reapproximated to the raw surface of the remaining major fragment. The use of a removable bolt suture has greatly facilitated the repair of major tendon ruptures, and produced more rapid and better results than any method previously employed.

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Arthrography of the Shoulder As a Diagnostic Aid in Tendon Injuries*

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THE symptom complex, pain and restriction of motion in the shoulder joint is thought to be common to disease or injury of one or more of the local subacromial soft tissue structures. The clinical picture is so similar and ill defined that, irrespective of which structure is thought to be basically at fault, there is no physical finding that consistently can be relied upon to characterize it.

The same patient examined by several competent physicians the same day may have his condition diagnosed differently by each. The most popular diagnoses are bursitis with tendonitis, capsulitis, bicipital tenosynovitis, adhesive capsulitis, supraspinatus syndrome, brachialgia and subluxation or dislocation of the biceps tendon less commonly used.

Treatment is empiric, and many cases respond satisfactorily. There are some, however, that are clinically indistinguishable from the others except that irrespective of the conservative treatment used, they improve either temporarily or not at all. It is with this latter group this paper is concerned.

More than twenty years ago Codman[1] emphasized that among this group there are many who have partial or complete tears of the rotator cuff mechanism. This diagnosis is considered by most clinicians as impossible to establish without surgical exploration.

CASE REPORTS

The following brief reports of two cases are cited as examples of the confusion that exists.

CASE I. N. P., a forty-seven year old man, received an injury to his right shoulder when the car in which he was sitting was struck in the rear end. The patient was thrown forward and used his right arm to protect his head from striking the windshield. Immediately afterward he had some discomfort in his right shoulder. During the next four months he consulted four physicians. Roentgenographic studies were made and diagnosed as normal. Three of the physicians made a diagnosis of bursitis; the fourth, of tendonitis. Treatment over the four-month period consisted of procaine and hydrocortone,® roentgen therapy (two series), diathermy, massage and Codman exercises. The patient experienced only temporary relief.

Examination four months after the aforementioned accident revealed atrophy of the deltoid, trapezius, supraspinatus and infraspinatus muscles. Crepitation was present bilaterally, but more marked on the right. The region of the greater tubercle of the humerus was extremely painful to palpation. Abduction, flexion, and internal and external rotation all were limited by pain and muscle spasm. However, when the painful regions were anesthetized with novocain® the patient had essentially a normal range of motion actively.

Inasmuch as the patient's condition had failed to improve after four months of conservative treatment, a tentative diagnosis of rupture of the shoulder cuff mechanism was made and surgical exploration of the shoulder joint recommended. This opinion was at such variance with the others that the patient had received that he requested another opinion. Subsequently he saw two nationally known competent orthopedists. One made a diagnosis of bursitis and recommended further roentgen therapy; the other did not make a diagnosis but recommended surgical exploration.

Operation revealed that the rotator cuff mechanism was detached over a region involving more than one-fourth of the circumference of the head of the humerus. This was repaired and the patient made an uneventful recovery.

CASE II. H. P., a forty year old woman, received a minor injury to her right shoulder. The following day she reported to the plant physician. X-rays were taken and reported normal. A diagnosis of bursitis was then made and during the next four months she saw two other physicians, both of whom concurred with the previous diagnosis and recommended conservative treatment. During this time the patient received two series of roentgen

* Funds for this investigation were provided by the Rockford Memorial Hospital Medical Foundation.

therapy, and on four occasions hydrocortone and novocain were injected into the shoulder joint region. She experienced only temporary relief.

Four months after injury, examination of the patient revealed atrophy of the deltoid, trapezius, supraspinatus and infraspinatus muscles. The region of the greater tubercle was moderately tender. Crepitation was present bilaterally, but more marked on the right. Active and passive abduction, flexion, and internal and external rotation were limited by pain and muscle spasm. Procaine was injected into the tender regions and after ten minutes the patient had essentially a normal range of motion actively.

A tentative diagnosis of rupture of the shoulder cuff mechanism was made and surgical exploration recommended.

The insurance carrier referred the patient to a well known competent orthopedist. This consultant stated that the patient did not have a rupture of the shoulder cuff mechanism and recommended further roentgen therapy.

The patient did not improve. Subsequently operation was performed, revealing a triangular-shaped tear in the shoulder cuff mechanism with a base approximately ½ inch in length and a perpendicular from the base to the apex of 1½-inches. The bursae were distended with fluid, and the biceps tendon was swollen, red and adherent to the bicipital groove.

From these two cases, which were seen within a period of only a few weeks, we drew the following conclusions: (1) Large tears of the rotator cuff mechanism are compatible with a normal or nearly normal range of motion if pain and muscle spasm are eliminated by the use of a local anesthetic. (2) Ruptures of the rotator cuff mechanism are more common than is generally appreciated and are difficult to recognize by the usual clinical tests. (3) A complete tear of the rotator cuff mechanism establishes a wide communication between the shoulder joint and the subacromial bursae. (4) An opaque medium injected into a shoulder joint in which such a communication exists should enter the subacromial bursae and thus establish the diagnosis.

Based on the latter assumption we began to make arthrographic studies of shoulder joints in 1953. A review of the medical literature reveals that others have reached the same conclusion. Two authors[2,3] report the use of contrast media in arthrographic studies of the shoulder, and another[4] mentions that he made arthrograms of the shoulder using oxygen. All agree that a communication between the shoulder joint and the subacromial bursae is abnormal and establishes the presence of a complete rupture of the shoulder cuff mechanism.

TECHNIC

Initial roentgenographic studies of the shoulder joint are made in anteroposterior projections with the humerus in internal and external rotation. The patient is then placed in a supine position on a roentgenoscopic table. The arm is supported so that it does not lie on the table but is in a neutral position at the patient's side. If the elbow is permitted to rest on the table, the humerus is in an extension position and the head of the humerus protrudes forward. This makes it difficult to insert a needle into the shoulder joint. The skin 1 inch below the acromioclavicular joint and just lateral to the coracoid process is anesthetized with procaine. A 3 inch No. 20 spinal needle is inserted into the shoulder joint under roentgenoscopic control. If the needle does not readily enter the shoulder joint, the forearm is flexed to 90 degrees at the elbow and moderate traction applied. This holds the head of the humerus away from the glenoid and increases the size of the joint space, thus facilitating the procedure. Joint fluid occasionally can be aspirated. If no joint fluid is obtained procaine is injected; and if there is no resistance to the injection, the needle usually is within the joint space. Twenty cubic centimeters of 35 per cent diodrast® and an equal amount of distilled water are thoroughly mixed and injected into the shoulder joint under roentgenoscopic control. The contrast medium dilates and fills the joint space. When either 40 cc. of the fluid have been injected or a definite resistance to further injection has been encountered, the needle is removed from the joint. Following careful manipulation of the shoulder, phototimed spot films are taken in the anteroposterior projections with the humerus in internal and external rotation.

A normal adult shoulder joint can hold 15 to 20 cc. of fluid without discomfort. If a tear in the shoulder cuff mechanism is present, 40 cc. or more can be injected without discomfort. There is little or no pain associated with the procedure, and there have been no complications. The diodrast solution is absorbed rapidly, leaving no detectable roentgenographic trace two hours later. Tissues removed from the shoulder joint at periods ranging from several

FIG. 1. Normal arthrogram, adult.

FIG. 2. Arthrogram of patient with rupture of the supraspinatus tendon.

days to over one year after arthrographic studies and then sent for microscopic examination are indistinguishable from similar tissues removed from shoulder joints not previously subjected to arthrographic studies. The ability to interpret arthrograms of this type is easily acquired. The variations from the normal are striking in most instances.

A normal arthrographic study usually will present the following characteristics (Fig. 1): (1) the capsule is elastic and ballooned out with opaque medium; (2) the volume capacity is equal to 15 to 20 cc. of diodrast; (3) the subscapularis bursae fill and are easily visualized; (4) the synovial sac extension into the biceps tendon groove fills with diodrast; and (5) the glenoid labrum is visualized as a region of lesser density.

If a tear in the shoulder cuff mechanism is present, the fluid pours out of the joint space into the subacromial bursae. Visualization of the subacromial bursae in this manner is diagnostic of this communication and indicates a tear in the shoulder cuff mechanism. (Fig. 2.)

Such a communication existed in thirty-two of the first seventy-two arthrographic studies we made. Of the thirty-two patients whose arthrographic studies were positive, twenty-four subsequently were operated upon. In twenty-three a large tear was found and repaired. The one positive arthrographic study, in which operation upon the shoulder joint demonstrated an intact shoulder cuff mechanism, has a possible explanation. Convulsions developed in this patient following a head injury. During one of the convulsive seizures he sustained an incomplete fracture of the greater tubercle of the humerus. The arthrographic studies revealed a communication between the joint and the subacromial bursae. It is possible that the opaque medium found its way out of the shoulder joint into the subacromial bursae through the fracture crevice.

Eight patients with a positive arthrographic study of the shoulder were not operated upon either because of age, physical condition or refusal. Six of these were traced and none had improved when last seen. Eight patients whose shoulder arthrographic studies revealed an intact cuff mechanism were operated upon for other reasons. In each instance the operative findings confirmed the arthrographic report. In three instances the findings warranted a diagnosis of incomplete rupture of the

supraspinatus tendon. (Fig. 3.) Subsequent operation confirmed the operative findings.

CONCLUSIONS

1. Arthrographic studies of the shoulder joint are a valuable aid in differentiating one of the causes of pain and restriction of motion in the shoulder joint.

2. An arthrogram provides a permanent record of an accurate and reliable method of demonstrating a complete or incomplete rupture of the shoulder cuff mechanism.

3. The technic of arthrography of the shoulder joint is simple to perform and the procedure is relatively painless for the patient.

4. The abnormal findings are striking and the ability to interpret the findings is readily acquired.

5. Safety of the procedure is attested to by the fact that there were no complications in the seventy-two cases reported herein and none are reported in the medical literature. Furthermore, tissues removed at operation and studied under a microscope showed no noteworthy changes, and no roentgenographic trace of the opaque medium remained two hours after intra-articular injection.

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DISCUSSION OF PAPERS BY DRS. WAGNER, McLAUGHLIN AND FRANCIS, AND KERNWEIN, SNEED, ROSEBERG AND ZEIER

Edgar L. Gilcreest (San Francisco, Calif.): As perhaps many of you realize, I have been interested in this subject of lesions of the biceps since 1924. In 1933 at a meeting of the American College of Surgeons I reported a collection of 100 cases, eight of which involved disinsertion of the biceps. As Dr. Wagner told you, from the reported cases at that time the injuries to the lower end of the biceps were only 3 per cent.

It is surprising after all these years to see that these patients are still neglected. Only recently I saw a patient who was injured in December, 1954 and had received physiotherapy until June, 1955.

Fig. 3. Arthrogram showing an incomplete tear in the supraspinatus tendon.

Finally, he was sent to a surgeon who operated on him. However, it was too late, with the result that the patient has a very weak arm.

This means much unnecessary money is being spent on cases of this kind. If they had had definitive treatment early, as Dr. Wagner has shown, much time and money would have been saved.

I like Dr. Wagner's operation because it copies nature very accurately. A permanent fixation is made, and good rotation, pronation and supination are obtained. So often the surgeon who is not interested in these cases merely sutures a portion of the tendon to the muscle. This leads to stress and strain, and pain for the patient.

The same holds true in the lesion of the upper end of the biceps when the disinserted end of the biceps is attached to the short head instead of being taken up and made a firm fixation on the coracoid process. When this is done the radius is attached to the shoulder girdle instead of the humerus.

I believe that this operation is a worth while contribution, and that when it is better known, it will be the operation of choice.

I also believe it is regrettable that insurance carriers do not recognize that these injured men should not receive physiotherapy for many months before they are sent to the surgeon because, with the passage of time, contracture and fibrosis occur and at best you obtain a very unsatisfactory result.

However, when these patients are seen early, it is often easy to get a 100 per cent result, as Dr. Wagner has demonstrated so clearly.

If desired, many of these patients can be operated upon under local anesthesia. More should be explored early. In this particular operation Dr. Wagner has shown us how he transects the recurrent radial artery, obtaining a good exposure and protecting the radial nerve.

Dr. Wagner has told me of a doctor friend of his who had this operation performed on himself. Unfortunately the radial nerve was sectioned, resulting in a radial palsy. This operation can be dangerous if it is not performed in the manner Dr. Wagner has shown.

Dr. Aitken told me that he has seen six cases of lesions of the biceps. This shows they are not as rare as they were when I began my study in 1933. We have gone a long way since then, and I think no group of surgeons is going to recognize and successfully treat these patients better than the Fellows of the American Association for the Surgery of Trauma.

Frederick Merwin Smith (New York, N. Y.): It seems to me that Dr. Kernwein's procedure is a most excellent test and one that may very well be compared with the gastrointestinal series and barium enema used when diagnosing soft tissue lesions deep in the body. Its safety factor seems to make it most valuable, especially since the diodrast disappears completely within a period of several hours, leaving no residual dye to cause irritation such as used to be noted when lipiodol was used in myelogram studies.

It is very difficult to diagnose accurately the condition of a shoulder after injury. Dr. Kernwein mentioned the fact that a great many of these patients are considered to have bursitis and are given physiotherapy of one sort or another for many months. This is not only a waste of time but also of very little benefit to the patient; it also causes extra and unnecessary expense. This test seems to go directly to the heart of the situation, and I believe that more of us should give it a trial. It is difficult even for doctors who examine shoulders frequently to make accurate diagnoses of cuff tears. I cannot do it, and most of the people I have been working with have a great deal of difficulty. In muscular individuals or people who are obese it is not only difficult to palpate the cuff region but also to put stress upon the various portions of the cuff to try to determine whether or not there is a possible tear in the supraspinatus, teres minor or subscapularis.

If we can perform a test with as little difficulty as Dr. Kernwein has shown, and with as great accuracy, I believe we are going to make many more accurate diagnoses and help a great many more patients by early operation.

James Keane Stack (Chicago, Ill.): When seeing these patients for the first time I am impressed with the relatively minor nature of the violence creating the injury.

I would like to ask Drs. Francis and Kernwein if a normal tendon ruptures under these circumstances?

Harrison Lloyd McLaughlin (closing): Dr. Kernwein's paper is a real contribution, and it seems likely that continued investigations along these lines will do much to clarify the many vexing and enigmatic problems attending treatment of the painful and disabled shoulder. By the use of contrast media a roentgen diagnosis of cuff rupture is now as reliable as gas under the diaphragm or a gastric filling defect. Most of the diagnoses made in this manner were followed by operative repair. I would submit that a diagnosis of cuff tear is not synonymous with the need for operation. Cuff ruptures exist in 20 to 30 per cent of all of the shoulders (from elderly persons) dissected in anatomy laboratories. Almost 50 per cent of patients presenting clinical evidence of a cuff tear eventuate with an eventually satisfactory shoulder without operation. Even when the diagnosis is certain, operative repair of a torn cuff is justified only by reason of adequate and persisting symptoms, and disability.

Joseph M. Janes (Rochester, Minn.): I believe Dr. Kernwein's method of diagnosis is very good.

We are accustomed to seeing arthrograms of the knee and the hip. Personally, I have had great difficulty in interpreting them.

As far as I know, this is the first presentation in America of the method of introducing diodrast into the shoulder joint. One might raise the question as to whether diodrast would be harmful to the joint. I do not think it would be more harmful in the joint than it would be in the arteries where it is being used daily.

The question also arises about sensitivity to diodrast. Certain people are sensitive and on rare occasions there have been serious reactions. I think that sensitivity could be determined by injecting a small amount intracutaneously into the forearm.

I hope to make use of this method to detect tears of the muscular tendinous cuff much earlier than I previously did.

Bernard E. McConville (Seattle, Wash.): I would certainly compliment Dr. Kernwein on this excellent procedure. I think anything that gives us an added adjunct to diagnosis is invaluable.

The question I would like to ask concerns the aftertreatment of surgical repair. In following a number of cases which have come to Washington Rehabilitation Center (after operation by various orthopedic men), it has been surprising to find that many patients have considerable disability.

I believe from the information I have received that the majority of the disability in cuff repair

has resulted from postoperative care. I believe that the abduction cast is a valuable adjunct in treating shoulder injuries.

I wonder what other men think of the use of a cast in the aftercare, in order to put the tendon in a more favorable mechanical position. I think Dr. Kernwein's work is very beneficial, and I hope to make considerable use of it.

STEPHEN SYLVESTER HUDACK (Cleveland, Ohio): I certainly do not wish to detract from Dr. Kernwein's excellent paper, but I would like to reinforce something that Dr. McLaughlin perhaps did not make clear, that is, the indications for operation should rest not only on x-ray findings but also on clinical findings.

It was my dubious fate to receive a supraspinatus tear in an automobile accident. I pondered for a long time whether or not something should be done about it, but I found that despite pain I had quite adequate motor power. I waited, and in about four months the pain disappeared.

I would appreciate Dr. Francis' comments on occult tears in the quadriceps mechanism. I have seen a few in which the surface of the quadriceps mechanism appeared entirely normal, but on careful exploration two-thirds of the deeper part of the quadriceps attaching to the patella had been pulled out. I think this kind of lesion can be overlooked if examined superficially.

H. MINOR NICHOLS (Portland, Ore.): In my experience treatment of disinsertion of the biceps has not been quite as easy as Dr. Wagner stated. The patients I have had have been quite old, with marked contracture of the muscles. Pain was a prominent complaint, apparently from scar tissue pulling on the median nerve. In these patients, because the excursion of the muscle was quite limited, I made a new insertion closer to the elbow joint, running a tendon graft through a hole drilled in the radius and weaving the graft into the stump of the tendon in the muscle after dissecting it out of the scar tissue around the nerve. Full and painless flexion and extension of the elbow, and full pronation and suppination of the forearm was attained. Strength was diminished.

I enjoyed Dr. Wagner's paper very much and I am anxious to try his method of fastening the tendon graft to the bone.

DR. EDGAR L. GILCREEST (San Francisco, Calif.): I wish to congratulate Dr. Kernwein on his splendid diagnostic ability. I think that his paper is going to be a real contribution in clarifying this very interesting subject about which we have been uncertain.

Dr. Kernwein referred to a dislocation of the long head of the biceps out of the inner tubercular sulcus. In 1936 I reported the first two patients operated upon for this condition, and I reviewed the literature since 1712, that being the year that Cowper reported the first cases. This article, published in the *Annals of Surgery*, contains the complete literature for over 200 years of all the cases of dislocation of the long head of the biceps. None of those patients were operated upon; they have been neglected for years and are still being neglected today. They are being treated for arthritis.

I wish to ask Dr. Kernwein what he did with the tendon of the long head of the biceps in the case he reported. I believe that the tendon should be taken from its insertion on the lip of the glenoid and transplanted up on the coracoid process, just as you would for a complete disinsertion or rupture of the long head. When this is done a fixation from the shoulder girdle to the radius results, and that is what is desired.

In some of the cases reported by Dr. Kernwein, I wonder if the lesion in the capsule of the shoulder was not partly due to a slow process of attrition. Many years ago, this was pointed out by Dr. Arthur Meyer, former Professor of Anatomy at Stanford University, who had observed it in the anatomic laboratory and reported a number of such cases. He found areas of attrition as big as a quarter or a dollar caused by the rotation of the head of the humerus under the acromion process.

An interesting question presents itself: what had caused this attrition of the capsule? It is believed that most of these men had worked daily over a period of years as painters or plasterers or vineyard pickers, using an overhead motion. I believe now with Dr. Kernwein's diagnostic method, we are going to explore more of these shoulders and repair them.

I wonder if in some of these cases part of the lesion had been previously caused by a process of attrition to which trauma had been added, such as some violent effort in lifting.

CARRUTH J. WAGNER (closing): Perhaps sometime I can show Dr. Nichols how much easier it is to re-attach the biceps brachii with a keyslot than it is to reattach it by slitting the tendon or drilling the radius and trying to get it through to the opposite side without disturbing important soft tissue.

I have not had occasion to re-explore a patient to see what the tendon looks like. I have used this same procedure many times in transferring the tibialis tendon in the foot. We have used it two or three times to repair ruptures of the Achilles tendon. I have re-explored several of these tendons. It is impossible to distinguish graft from tendon. It assumes the size and shape necessary for the function of the part as do tendon grafts in the hand. For example, we can take the palmaris longus, put it in the finger and examine it in a year, and it will be two or three times the size it originally was. The same occurs when fascia lata is used as a graft.

Again, any dissection about the radius is going to increase the complications; in using the graft in the keyslot the bicipital tubercle exposes itself and no dissection is necessary.

KENNETH C. FRANCIS (closing): In answer to Dr. Stack's question, does the normal tendon rupture, in this series of cases almost every patient showed evidence of attritional degeneration of the tendon fibers at operation. In a few cases of fracture of the patella in young people, when the rupture force that fractured the patella continued, a normal quadricep tendon was torn.

In answer to Dr. Hudack's question, I believe there were several cases in which there was an intact layer of the external capsule after fracture of the patella. However, all patients in the series demonstrated inability to extend the knee against gravity.

GRAHAM A. KERNWEIN (closing): Dr. Stack has asked whether we believe a normal tendon will rupture. We do not believe it will, and this is based on two facts. First, there has never been a rupture of a shoulder cuff mechanism reported in a patient under the age of thirty-two which would indicate that age and trauma play a part. Second, experimental work on the rupture of tendons done by McMasters revealed that a normal tendon is the strongest link in the muscle-tendon-bone chain. He found that a tendon will avulse from the bone or rupture at the musculotendinous junction before the tendon will rupture.

We may assume, therefore, from experimental and clinical evidence that when a tendon ruptures it is diseased, or otherwise it would be avulsed or rupture at the musculotendinous junction.

In answer to the question raised by Dr. McLaughlin and Dr. Hudack, "should all of these patients be operated upon?", may I say that there were eight patients whom we reported and so stated that they were not operated upon either because they refused or because we believed that their age or symptoms did not warrant it. I should like to cite two such examples.

The first patient was seen approximately one year after he had been operated upon. He stated that he had occasional pain in the opposite shoulder. He was interested in knowing whether or not something was wrong with this left shoulder. An arthrogram revealed a communication between the shoulder joint and the subacromial bursae. As this was his left arm and it bothered him only occasionally, both he and I agreed that the symptoms were insufficient to warrant operation. A second case, a banker weighing approximately 300 pounds, was referred to me because of continued pain and restriction of motion in the shoulder. He was being treated elsewhere with the usual conservative methods. His chief concern, as he expressed it to me, was whether or not he had arthritis or cancer. When an arthrogram was performed and the results explained to him, he understood the cause of his complaints and was satisfied. His overweight, and the fact that his shoulder bothered him very little in the course of his normal duties, were the deciding factors against an operation.

The test, as we use it, is performed only for diagnostic purposes and to aid the patient in an understanding of the cause of his complaints. The question of whether or not he should be operated upon is one of surgical judgement.

One must, of course, recognize that the patient is in a doctor's office because he has a complaint and is concerned about it. If, by means of an arthrogram, a logical explanation can be given the patient, that may be all that is desired or indicated. Actually the plan that we have is to explain to the patient that his discomfort is perhaps the most outstanding reason for operation. We tell him that we liken it to the Republican slogan, "When have you had enough?" If he has had enough, I think he should be operated upon.

I wish to thank Dr. Janes for his kind remarks regarding the usefulness of the test. Also, I appreciate his suggestion that an intracutaneous injection is indicated to rule out the possibility of sensitivity to the drug. We have not done this but I believe it is a fine suggestion.

In reply to Dr. McConville as to the method of our postoperative fixation, may I say that initially we repaired the tear and then left the patient with his arm in a sling at his side, as recommended by Dr. McLaughlin in his article dealing with the transacromial approach. Because we encountered great difficulty in mobilizing these shoulders, and particularly in gaining abduction, we discontinued this method. Instead, we now put on a shoulder spica cast at the time the operation is finished. The arm is held in approximately 60 degrees of abduction and 20 degrees of flexion. The cast is worn for three weeks. The top of the arm portion of the cast is then removed and motion is started from the 60 degrees of abduction upward. Once the patient can actively abduct his arm out of the cast to 90 degrees and hold it, the cast is removed. Progress with this regimen has been more rapid. The patients will improve over a period of eighteen months at least.

In reply to Dr. Gilcreest's remarks about the dislocation of the long head of the biceps muscle, may I say that we have had eight such patients. In each the biceps tendon was completely dislocated. We have treated some patients by attaching the tendon to the coracoid process. Recently, however, we have removed the intra-articular portion and after raising a portion of the bony floor of the bicipital groove, fastened the tendon with a staple. This method has proved entirely satisfactory in fixing the tendon. We also believe it is much more simple.

In a number of cases we found the tendon to be red with many adhesions between it and the surrounding portions of the bicipital groove. This was the picture that has been described as characteristic of bicipital tendonitis and is supposedly responsible for pain. We intentionally left the bursae and the tendon with its adhesions, and closed only the capsule. The postoperative course was indistinguishable from others. From this we concluded that tendonitis was secondary and the adhesions have not altered the subsequent course.

A negative arthrogram also may be valuable. Recently we saw a woman in consultation with a very fine surgeon. She had a typical history of an injury adequate to rupture the shoulder cuff mechanism. Upon examination she could not abduct or forward flex the arm. We anesthetized the painful areas about the shoulder and following this she exhibited no increase in ability to abduct or flex the arm. By the usual standards this woman would have been considered to have a rupture of the shoulder cuff mechanism. An arthrogram was performed and revealed an intact shoulder cuff. This arthrogram, along with a normal arthrogram of another patient and an abnormal one, were reviewed with the patient and her surgeon. She was an intelligent woman and seemed to appreciate that we had very definite evidence that there was no alteration in the muscles or tendons involved. She was taken to physical therapy, where after approximately one hour she was able to put her arm through a normal range of motion. We believe that the concrete evidence presented by the arthrogram in this instance convinced both the patient and the surgeon that an operation was not required. We also believe the evidence was directly responsible for her rapid response to conservative treatment.

Secondary Restoration of Finger Flexion by Digital Tendon Grafts

AN EVALUATION OF SEVENTY-SIX CASES

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IN the repair of lacerated flexor tendons *in situ*, the most frequent failures are encountered when the laceration occurs between the distal palmar crease and the distal finger joints. The secondary repair of functional defects resulting from injury sustained in this "danger zone," or "no man's land,"[1] is best done by tendon grafting. This report offers an analysis of the results obtained in seventy-six tendon grafts to the digits, all of which were performed six weeks or more following injury.

For an explanation as to why tendon repair is so often unsuccessful in this "danger zone," we have but to review the anatomy of the hand and consider its dynamics of function. The flexor tendons are large, strong cables of smoothly compressed fibrous tissue which transmit the force of powerful muscle motors located in the forearm. Since there are three phalanges in the fingers and two in the thumb, it is possible to obtain movement by virtue of joint motion. Thus a profundus tendon can produce flexion of the distal phalanx through three-quarters of a circle or 270 degrees. For the hand to grasp smoothly and hold firmly it is essential that the transmission of force be accomplished without friction and that the points of leverage afford maximum utilization of power. Leverage is derived from the fulcrum points of pulleys or, in the fingers, the fibrous retaining ligaments. Tendon pull is thus transmitted to the phalanges which flex at the metacarpophalangeal and interphalangeal joints. With this resultant change of direction of pull with finger flexion, gliding motion of the tendons is provided by the tendon sheaths. A tendon sheath is not unlike the peritoneum or pleura in that it is a mesodermal envelope with a parietal layer and a reflected visceral layer. Between these two layers of moist slippery tissue, friction of movement is minimized. Since the parietal layer of the sheath and the retaining ligamentous structures blend so intimately together, we can consider them as a single unit for practical purposes. A tendon sheath with its ligaments may then be described as a fluid-bearing mechanism of gliding motion, existing where there is change in direction of pull. (Fig. 1.)

If a lacerated flexor tendon is repaired within a sheath, there will be resultant scar about the healed wound and at the site of tendon anastomosis. Frequently this will prevent the restoration of gliding motion because of adherence of scar. If motion is established, a nodulation will exist at the point of juncture which will impinge at the pulley. (Fig. 2.) This may result in irritation and subsequent tendon fixation or separation.

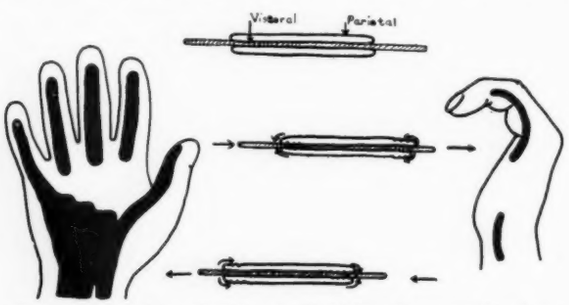

FIG. 1. Tendon sheath. A fluid-bearing mechanism of gliding motion existing where there is change in direction of pull.

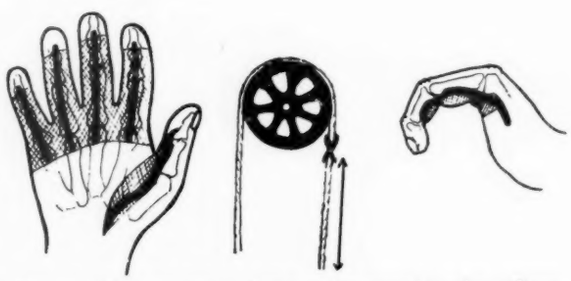

FIG. 2. Danger zones for tendon repair exist where there is change in direction of pull.

TENDON GRAFTING

Indications. The indication for tendon grafting is inability to flex adequately the interphalangeal joints following injury in the "danger zone" providing, of course, that some simpler procedure would not suffice. The decision to operate will then depend upon the time elapsed since injury, concomitant injuries, complications following injury, the condition of the hand and the willingness of the patient to cooperate.

Technic. In the secondary repair of digital flexor tendons it is advisable to remove the sublimis tendon from the finger and palm, up to its muscular origin just above the wrist. One of the distal stumps of the sublimis tendon, which inserts on the proximal portion of the middle phalanx, should be left long enough to bridge the proximal interphalangeal joint to prevent hyperextension. If there is ample cicatrix to prevent hyperextension, the stumps may be cut short and their ends buried in soft tissue. In any event the unsatisfied raw ends of tendon should not be left to adhere to the newly reconstructed tendon.

The sublimis tendon thus removed may be used as a graft to restore the continuity of the profundus tendon; however, the sublimi of the index, middle and ring fingers are usually too great in diameter to serve as grafts. Heavy tendons such as these are likely to undergo considerable central necrosis and swelling, with subsequent adherence to the surrounding structures. The palmaris longus tendon serves as an excellent graft and is present in approximately 87 per cent of humans. This tendon is small in caliber, somewhat flat, easily accessible, covered with good paratenon and usually long enough. If the palmaris is not present or if more than one tendon is needed, the long extensor tendons of the second, third or fourth toes can be used. These tendons are also flat, small and long enough, and their removal does not alter function of the foot. The long extensors of the great toe and the little toe should not be used since the former is essential to normal gait and the latter is the only extensor to the little toe. The second, third and fourth toes possess intrinsic short extensors that will suffice for normal toe function. While the extensor tendons of the toes are quite satisfactory as grafts, they are still somewhat small and tend to fray when sutured. In addition, an incision in the foot usually prolongs anesthesia time, hospitalization and gross disability. The palmaris longus tendon is the graft of choice. In obtaining tendons for transplantation, careful dissection is imperative; they should never be "pulled out." Rather, the exposure should be generous and the graft obtained by sharp dissection throughout its length so that paratenon will remain on the surface of the tendon. There is usually considerable disparity between diameter of graft and flexor tendon to be restored. This is probably advantageous during the healing period for it permits ample space under the ligaments in which the graft can swell. If the tendon functions properly, hypertrophy will subsequently occur in proportion to the demand.

The graft, when placed in the finger, should be passed under the ligaments. If the ligaments have been damaged by injury, infection or fibrosis so that repair is impossible, a new pulley should be formed by passing a piece of small flat tendon graft around the phalanx. This dissection should be done carefully to avoid injury to the extensor mechanism. Pulleys are essential to prevent bowstringing of the tendons and to maintain the proper efficiency of flexion.

Distally the graft should be fixed at the insertion of the profundus tendon either by suture to the tendon stump or by fixation to bone. The method of suture is optional. The author has usually employed the Bunnell "pull-out" wire suture[1] distally, fixing the graft to the profundus stump without drilling the distal phalanx. The proximal anastomosis of the graft to the profundus is best done just distal to the origin of the lumbrical muscles. These muscles which move with the profundus tendons can be loosely wrapped about the anastomosis to prevent adherence. The suture material at this anastomosis may be silk or pull-out wire. Tension on the tendon is adjusted so that the finger lies in proper relation to its fellows, when the wrist is slightly extended.

Analysis of Results. This series of seventy-six tendon grafts is not large enough to afford critical statistical evaluation; however, certain generalizations appear to be significant. It is anticipated that this study will be continued, and as such it should afford guidance in the future. Since all of the tendon grafts in this series have been performed or intimately supervised by one individual, this should

eliminate some measure of variation. This study covers a period of six years, during which time aptitudes and technics were being developed and case selection has become more critical. With the passage of time there has been a continuous improvement in results.

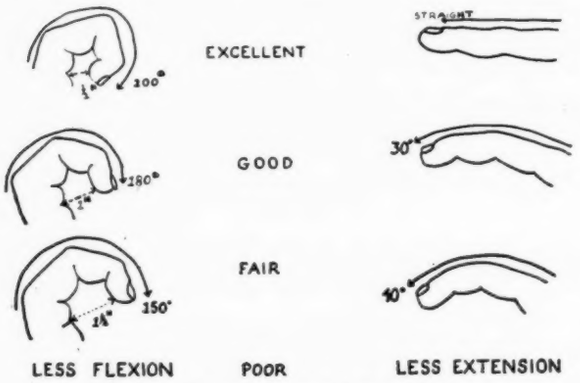

FIG. 3. Results in tendon grafting.

The seventy-six digital tendon grafts have been performed in sixty-two patients. In ten patients the grafts were multiple, and repeat grafts were done in four cases. All the tendon grafts were secondary to injury in the sheath area and were done six weeks or more subsequent to injury. In this series failures in tendon grafting have been included as poor or fair results and the repeat grafting has been considered as a separate tendon graft procedure. In the four failures there was tendon separation at the proximal anastomosis in two cases, tendon adherence in one and too short a graft in another.

In any attempt to evaluate the end results of tendon grafts in "no man's land," the basis of evaluation must be a mechanical measure. It is quite impossible to determine functional return since the demands of function are so variable. As an example, the range of flexion of the ring finger that may be satisfactory for a carpenter may be incapacitating to a violinist.

In 1950 Boyes[2] presented a method of evaluation of end results in digital flexor tendon grafts. His criterion was based on the measured distance that a tendon-grafted digit lacked in flexing the tip of the finger to the distal palmar crease. He classified the end results of this measured distance as lacking "½ inch," "1 inch" and "1½ inches." This measure does not take into consideration the degree of active extension of the digit. By this evaluation it is conceivable that a finger with ankylosed interphalangeal joints could be considered a good result since the fingertip might flex well into the palm. In addition, this evaluation does not relate the range of motion at the finger joints. The effective range of motion at any joint is the difference between flexion and extension. To determine excellence or the lack of it in finger flexion, proportionate values must be assigned to the composite flexion and extension possible at the metacarpophalangeal and interphalangeal joints.

This study has been based on an evaluation of the factors of composite digital extension and flexion plus the measured distance of the lack of flexion of the fingertip to the distal palmar crease. (Fig. 3.) The classification is as follows: *Excellent:* Ability to extend the finger fully, to attain composite flexion of 200 degrees and flex the pulp within ½ inch of the distal palmar crease. *Good:* Ability to extend the finger to within 30 degrees of straight, to attain composite flexion of 180 degrees and flex the pulp within 1 inch of the distal palmar crease. *Fair:* Ability to extend the finger within 40 degrees of straight, to attain composite flexion of 150 degrees and flex the pulp within 1½ inches of the distal palmar crease. *Poor:* Less than fair.

Since the results of tendon grafts of the thumb cannot be measured by these standards and since there is so much variation in degree of normal flexion of the thumb interphalangeal joint, the following scheme has been used. Flexion is compared by per cent with the range of motion at the interphalangeal joint of the thumb of the opposite hand. Seventy per cent or more is considered excellent; 60 to 70 per cent, good; 40 to 60 per cent, fair; and less than 40 per cent, poor.

Distribution. The distribution of tendon grafts was thirty in the right hand and forty-six in the left hand. The index finger was grafted thirty times, the middle finger fourteen, the ring finger twelve, the little finger eleven and the thumb nine. The predominance of tendon grafts to the index finger is probably due to the more troublesome nature of the functional defect in this principal digit. The best results were obtained in the thumb grafts.

General Factors. Age: Patients under thirty years of age and over ten obtained the best end results. Children less than five are not considered good candidates for tendon grafting because of lack of cooperation.

Sex: In this series there were fifteen females and forty-eight males. Slightly better results were obtained in the former than in the latter. This may be due to the fact that generally women have more supple hands than men.

Body type: Gross evaluation of body type as to height and weight was not found to be of any significance in the analysis of results in digital tendon grafts.

TABLE I
POSTOPERATIVE COOPERATION

	Excellent	Good	Fair	Poor
Cooperative.....	20	15	11	13
Uncooperative.....	2	2	4	9

Hand type: Hands were classified as to length and width. It was found that those patients with thin, long, pliable hands obtained somewhat better results with tendon grafting than those with rigid, broad, coarse hands.

Intelligence: The evaluation of intelligence was based upon the impressions of the author. This evaluation indicated that more intelligent individuals obtained slightly better end results. The difference, however, was not as great as might be expected.

Adjustment: There was a relationship between the reaction of patients to their functional defects and the eventual functional result of tendon grafting that they attained. Those with the mature attitude of annoyance with the disability obtained better results than other groups. Those who displayed the mature attitude of acceptance did somewhat better than those who appeared immature. Immaturity was judged by rejection, fixation, passivity or timidity. However, the mere fact that these patients subjected themselves to surgical correction indicates a mature desire for improvement.

Postoperative cooperation: The willingness of the patient to participate in his rehabilitation was a significant finding. Usually it reflected the desire of the patient for improvement. Those individuals who cooperated fully in their efforts to obtain the best result possible and those who cooperated to the point of adequate functional return were able to secure satisfactory (excellent or good) results in 59 per cent, while uncooperative patients obtained only 23 per cent satisfactory results. It might be expected that this difference would be greater. (Table I.)

Local Factors. Skin loss: Loss of skin coverage was a factor in ten instances. In six of these the result was poor, in three fair and in one good. While this grouping is small, it is evident that skin loss constitutes a major problem in tendon grafting.

TABLE II
EFFECT OF PRIMARY TREATMENT UPON THE END RESULTS IN DIGITAL FLEXOR TENDON GRAFTING

Primary Treatment	Excellent	Good	Fair	Poor
None.....	8	2	4	7
Skin suture.....	5	8	4	5
Tendon repair.....	9	6	6	10
Tendon graft.....	..	1	1	..

Primary treatment: The type of surgical management of the acute injury was not as significant in this evaluation as might be expected. Slightly better results were obtained if no surgical correction was attempted or if only the skin was repaired. Primary tendon anastomosis had been done in thirty-one digits, all of which failed as manifested by secondary tendon reconstruction. In this group of thirty-one the eventual end result with tendon grafting was satisfactory (excellent or good) in fifteen. (Table II.)

Single or multiple tendon injuries: These factors appear to be significant in this analysis. Patients with single tendon injuries obtained 64 per cent satisfactory results, while only 15 per cent of those with multiple tendon injuries could be classified as satisfactory.

Associated injuries of nerve, bone and joint: Compound injuries were present in thirty-five instances. Generally, nerve injuries were not significant to the eventual result of tendon function; however, among twelve patients with associated bone and joint injuries, there was one good result, seven fair results and four poor results. Bone and joint injury detracts from the eventual functional restoration.

Primary complications: Disturbances in healing associated with the original injury in the form of enduration, drainage or frank infection reduced the incidence of good tendon graft results. In thirty-six cases in which there was

no complication, 69 per cent had a satisfactory result, while among forty in which complications had occurred, only 35 per cent obtained satisfactory results. With any history of infection, tendon grafting was deferred for at least three months after the infection had subsided.

TABLE III
RESULTS IN SEVENTY-SIX DIGITAL FLEXOR TENDON GRAFTS

Condition of Hand (preop.)	Excellent	Good	Fair	Poor	Totals
Supple.....	16	6	3	3	28
Scarred.....	5	8	11	3	27
Stiff.....	1	3	1	10	15
Congealed...	..	..	..	6	6
Totals....	22	17	15	22	76

Interval operations: Each surgical procedure performed on the injured part between the time of injury and tendon grafting appears to reduce the possibility of eventual success. Among the twenty-four patients who had interval operations, only seven (29 per cent) obtained excellent or good results.

Lapse of time: The period of time between injury and tendon grafting was not significant in this series. Two tendon grafts were done at six weeks, all others after two months or more.

Preoperative condition of the hand: The most significant factor evaluated in this study is related to the preoperative condition of the hand. This emphasizes the necessity for critical evaluation of any prospective candidate for tendon grafting. The following classification has been found useful in the selection of cases: Group 1: "The supple hand"—minimal scarring; Group 2: "The scarred hand"—midline volar digital scar or other limiting cicatrix; Group 3: "The stiff hand"—cicatrix plus intrinsic restriction of joint motion; and Group 4: "The congealed hand"—fixation by extensive cicatrix of soft tissue and joints.

Of the twenty-eight "supple hands," 79 per cent obtained excellent or good end results. Of the forty-eight hands in which scar was a factor, only 35 per cent secured excellent or good end results. (Table III.)

Technical Factors. Operative time: Excessive operative time evidently endangers the eventual result in tendon grafting. More than three hours is considered excessive.

Tourniquet time: Prolonged use of the tourniquet, like prolonged operative time, is associated with poor end results. Prolonged use of the tourniquet unquestionably interferes with the normal process of repair. Results were better if the tourniquet time was less than two hours.

Source of tendon grafts: The sublimis was used eight times, the palmaris longus thirty-one times and the extensor tendons of the toes thirty-seven times. Considerably better results were obtained with the palmaris longus than with the long extensor tendons of the toes. The palmaris longus was used thirty-one times and gave satisfactory results in twenty-six instances (84 per cent). Sublimis tendons were used as grafts eight times and only three were satisfactory (37.5 per cent).

Pulley construction: Ligament reconstruction was necessary in fifteen cases, five of which obtained satisfactory (excellent or good) end results. In three patients, it was necessary to divide or remove the new pulley mechanism to free the tendon graft.

Insertion of slippery substance: This was done six times, with satisfactory results obtained only twice.

The sublimis tendon: This tendon was removed in sixty-two of the sixty-seven tendon grafts to the fingers. In five instances it was possible to preserve the sublimis tendon, since injury had been sustained distal to this structure. Among these five cases the results were excellent in three, good in one and poor in one. The sublimis can only be retained in highly selected cases when all other conditions are optimal.

The type of suture material: Essentially the same suture material, wire and silk, was used in all instances.

The method of tendon suture: Pull-out wire sutures were used distally and silk sutures proximally.

Multiple procedures: Concomitant reconstruction at the time of tendon grafting such as nerve repair, other tendon grafts, pulley reconstruction or tendon transfers decreased the incidence of good end results.

Postoperative Factors. Complications: In this series there were ten wound complications. Five patients had cellulitis, one patient had drainage and four had frank infection; all had poor results. Tendon grafting was repeated in one of the cases with infection.

TABLE IV

	Excellent	Good	Fair	Poor	Totals
Mature Cooperative Supple hand	15	4	3	..	22 { 68% Excellent; 86% Excellent or good
Immature Uncooperative Scarred, stiff or congealed hands	7	13	12	22	54 { 13% Excellent; 37% Excellent or good

Period of immobilization: In all seventy-six cases the period of immobilization was three and a half weeks, or twenty-five days.

Early status of tendon function: With the removal of the splint, early motion was limited by stiffness; however, in nine instances the tendon seemed fixed. A fair result was obtained in three of the nine, the remainder being classified as poor. In four cases the tendon separated proximally. In spite of this, one had a fair range of motion; the other three results were poor. Two of the four separated tendons were regrafted with excellent and poor results, respectively.

Rebabilitation. When the bandage and splint were removed at three and a half weeks, the patients were instructed to bathe the hand in warm, soapy water several times each day. Bathing was followed by massage with any bland ointment, care being used to employ very little grease. They were instructed that the joints and tendons should not be stretched passively but that gentle active motion should be undertaken. The patients were advised that the hand would swell slightly. When the patients returned in one week they were instructed to continue the aforementioned program but to undertake active use of the part. Light work was encouraged. The following week heavy work was advised and the patients were advised to return to work and full activity. Gradual improvement was subsequently noted for six months or more. Physical therapy was not prescribed for it is believed that the patient is his own best therapist.

Range of motion: The best end result of composite range of motion was 263 degrees. Eight patients now have a range of motion exceeding 250 degrees.

COMMENTS

It appears that some of these multiple, variable factors are especially significant in relation to the end results of secondary tendon grafting. The following factors include those which have been shown to be most significant: (1) Age, (2) intelligence, (3) adjustment, (4) cooperation, (5) single or multiple tendon injuries, (6) skin loss, (7) associated injuries, (8) primary complications, (9) interval operations and (10) condition of the hand.

If a composite evaluation of maturity, cooperation and condition of the hand is made, it is possible to predict end results in a useful manner. (Table IV.)

No attempt has been made to evaluate the relationship between the various factors. Certain groups of factors are obviously related, such as multiple tendon injuries, skin loss, associated injuries, complications and condition of the hand. Obviously such problems require prolonged surgical procedures under circumstances that are not ideal for tendon grafting. These patients are most likely to have poor results. On the other hand, patients with single tendon injuries, good intelligence, adjustment and cooperation, and without skin loss or other associated injuries who possess a supple hand and a strong available palmaris longus, present the ideal arrangement for restoration of function.

This evaluation of results is on the basis of anatomic and mechanical features, for it is impossible to establish degrees of functional return. Unquestionably most of these individuals who have fair or poor results have derived definite functional benefit. Therefore, we cannot select these patients on the basis of possible anatomic restoration but must use tendon grafting wherever it would seem beneficial. There should be a word of caution, however, in that a tendon graft is an elaborate procedure and requires a prolonged period of inactivity. Careful criteria of selection should

be used, for the benefit obtained must compensate for the effort and time expended.

SUMMARY

A series of seventy-six secondary flexor tendon grafts to the fingers have been presented for evaluation. In all instances the initial disability had resulted from injury in "no man's land." This evaluation indicates that cooperation of the patient and the preoperative condition of the hand are the most significant factors in predicting results.

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Treatment of Ruptured Tendons of the Hand and Wrist

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THE evidence of ruptured tendons in the hand is very low. In fact, these injuries are so rare that when the occasional case is seen, the diagnosis is often not readily made. Because of this fact and because in the ruptured tendons that we have seen a diagnosis had not been easily made, it was decided to review the ruptured tendons of the hand and wrist we have encountered in the past five years.

Normal tendon itself does not rupture through its substance. There is usually some etiologic factor that causes a rupture to occur. This can be a severe sudden force to the hand, thumb or fingers causing a rupture of the tendon at its insertion or a tearing of the tendon from its muscle belly. In addition, fractured bones, such as a fracture of the distal end of the radius, may act upon the tendons going over them to cause rupture. In the past various types of disease, such as tuberculous tenosynovitis, gonorrhea, syphilis, arthritis and so on, have been thought to be an etiologic factor.

It has been stated that in most cases of ruptured tendons of the hand and wrist the joints subluxate, the bones are fractured and the muscles are torn through at the musculotendinous junction.

Experimentally, it has been shown in healthy adult rabbits that when normal tendon is subject to severe strain, either the tendon insertion was pulled away, the muscle belly ruptured, the musculotendinous junction separated, the muscle origin pulled out, or fracture of bones occurred. The tendon or muscle, in pulling away from a bony insertion or origin, usually detaches a small fragment of bone.[1]

Adams[2] reports the case of a seaman who caught the right middle and ring fingers in a door and apparently pulled the hand away quickly. There was a crushing injury to the ring finger and amputation of the distal phalanx of the middle finger. The tendon of the flexor digitorum profundus and a portion of the muscle belly was pulled away with the amputated stump.

Last[3] reports on two digits, specimens from the Hunterian Collection, which were avulsed by being caught in revolving machinery. One is the ring finger of a twenty-six year old man. The tendon of the flexor digitorum profundus is completely avulsed from the muscle. The flexor digitorum sublimis and the extensor digitorum communis tendons have ruptured at their insertions. The other digit is a thumb of a girl. The extensor pollicis longus and the flexor pollicis longus tendons are both avulsed from their muscle bellies and remain with the amputated stump.

Spontaneous tendon rupture or rupture through tendon substance may occur in a diseased or previously injured tendon. It has been shown that obstruction to the blood supply of 1 cm. of a normal tendon by double ligation caused rupture in this obstructed area as early as four weeks afterward when it was subjected to strain.[1] A case of tendon rupture due to gonorrhea tenovaginitis has been reported.[4] Mason,[5] quoting from Kanavel's files, found ten instances of rupture or impending rupture of tendon of twenty-one available records of tuberculous tenosynovitis. Typhus fever, syphilis, gout or deforming arthritis may be a cause of tendon rupture.[5] In addition, it has been suggested that tumors of tendons may be a cause for rupture although no cases have been reported.[6] Typhoid fever may also be a cause of tendon or muscle rupture. Haldeman and Soto-Hall[7] classify muscle and tendon injuries based on causative forces and contributing factors as follows:

I. Direct trauma
   A. A blow or crushing force applied to a muscle or its tendon
II. Indirect trauma
   A. A stretching force applied to a contracting muscle or its tendon
   B. A sudden or unusually forceful contraction of a muscle
III. Spontaneous rupture
   A. Post-traumatic
      1. A single injury which so weakens a muscle or a tendon that rupture occurs at a later date
      2. Often repeated minor injuries; for example, the rupture of the extensor pollicis longus tendon following Colles' fracture
   B. Disease of the tendon or muscle: tuberculosis, gonorrhea, syphilis or typhoid
   C. Senility, a factor that is often combined with one of the foregoing conditions
IV. Dislocation of tendons
V. Herniation of muscle through its sheath

EXTENSOR TENDONS

Detachment of the extensor tendons at their insertion into the distal phalanx is the most frequent type of tendon rupture. Here indirect injury may lead to rupture of normal and pathologic tendons; the normal tendons most frequently ruptured are the extensors of the middle, ring and little fingers at their insertion into the distal phalanx. This injury is not infrequently seen on the baseball field, due to a blow on the tip of the extended finger, thus the term "baseball finger." The condition has also been called "mallet finger," due to the typical deformity.[8] Men are more frequently affected than women and the injury occurs more often in digits of the right hand. The usual history is that of a blow on the tip of an actively extended finger after which the patient cannot actively extend the distal phalanx of the injured finger. The injury consists usually of a capsular tear and separation of the tendon from its insertion. Lateral x-rays may show a small shell of bone pulled away with the tendon insertion. Swelling and pain appear soon after injury and ecchymosis may be present. The acute injury may be mistaken for dislocation of the distal phalanx and the finger placed on a splint, the true nature of the injury becoming evident only when the splint is removed and the typical deformity seen. This deformity is due to the unopposed pull of the flexor digitorum profundus.

In the fresh injury, splinting with the distal interphalangeal joint in hyperextension and the proximal interphalangeal joint in flexion may occasionally produce good results. However, the tendon may heal in a lengthened condition with some residual deformity; and since the joint capsule is always injured with the possibility of tags of tissue lying within the joint which may become ankylosed, immediate operative repair of the injury is the treatment of choice. The skin incision is made in a modified S-shape: the long arm on the lateral aspect of the middle phalanx; the short arm crossing the finger transversely in the skin crease over the distal interphalangeal joint and then extending distally along the lateral aspect of the distal phalanx as far as is necessary to expose the site of attachment of the extensor insertion on the distal phalanx. The tendon and joint capsule are then carefully approximated with fine non-absorbable sutures of silk or cotton with the finger held in hyperextension. If the tendon is avulsed from the bone, the periosteum over the base of the distal phalanx is curetted down to raw bone and the tendon sutured to the bone with fine cotton, silk or wire sutures.

If primary treatment is unsuccessful or if the patient is seen late, a reconstructive procedure is necessary to correct the deformity. Secondary tenorrhaphy may be done. A portion of the scarred tendon can be excised and the ends approximated carefully, or the scarred portion can be overlapped. A tendon graft anchored to normal tendon and bridging the weak area may be utilized. The graft may be a split portion of the palmaris longus or of the long extensor tendon to the second great toe 3 or 4 inches long and about the thickness of No. 1 catgut.[8]

The tendon graft is first anchored at the insertion, being passed through the distal stump of the tendon or through a side-to-side drill hole in the base of the distal phalanx. It is then passed back across the joint, threaded through the scarred area and into sound tendon proximal to the tear. Tension on the graft is adjusted in such a way that the joints are flexed to the same degree as those of the fingers on each side when the wrist is flexed or extended. The graft is then sutured in place with fine non-absorbable sutures. Following operative repair of the extensor tendon, the finger is kept in hyper-

extension with a volar splint for three weeks after which motion is allowed.

In our series of cases we encountered a number of ruptures of the extensor tendon at the distal interphalangeal joint. On the whole, treatment has been quite satisfactory, especially in the secondary procedures that were carried out. Tendon grafting procedures have been done with some improvement but not complete return of function. More recently we have done the secondary suture on the extensor tendon at its insertion and then a small Kirschner wire has been placed intramedullarly through the distal phalanx into the middle phalanx. This provides a certain amount of internal fixation so that it is not necessary to apply the splint maintaining the finger in complete extension. The splint we use is a tongue blade splint built up on the end which provides extension of the distal phalanx itself. We have tried the method of flexing the proximal interphalangeal joint and extending the distal joint but find that the position is awkward to most people, and it is only by applying the other type of extension splint that we are able to obtain good results. The Kirschner wire is removed at the end of four to six weeks and usually results in definite improved function. In long-standing cases it is probably better to perform arthrodesis rather than try secondary tenorrhaphy.

A much less frequent type of tendon rupture is that of the extensor tendon over the middle interphalangeal joint. The extensor tendon forms an expansion or aponeurosis over the proximal interphalangeal joint made up of two lateral slips which, with fibers from the tendons of the lumbricales and interossei, pass on to insert into the base of the distal phalanx and a central slip which inserts into the base of the middle phalanx. Injury here usually consists of rupture of the central slip at its insertion into the base of the distal phalanx and is produced by sudden passive flexion of an actively extended finger at the proximal interphalangeal joint. This injury is commonly called "buttonhole" rupture since the knuckle, on flexion of the finger, protrudes through the two lateral extensor tendon slips as a button through a buttonhole.[9] The typical deformity produced by this injury is that of hyperextension of the distal phalanx and flexion at the proximal interphalangeal joint on attempting to actively extend the finger. Diagnosis is made on the history of injury followed by pain and swelling at the proximal interphalangeal joint and the finding of the typical deformity.

Splinting with the involved finger in extension as a method of treatment in fresh cases has been tried, but the results are usually poor.

Operative repair is the procedure of choice. A modified S-shaped incision over the proximal interphalangeal joint with suture of the proximal portion of the central slip of the extensor tendon to the base of the middle phalanx with fine non-absorbable suture may give good results.

When the patient is seen late or when primary repair has failed after injury of the "buttonhole" type, a tendon graft is necessary to correct the deformity. A graft from the palmaris longus tendon or long extensor tendon to the second toe, about the diameter of No. 1 catgut, is taken. It is placed through the distal stump of the central slip of the extensor tendon or through a side-to-side drill hole in the base of the middle phalanx, carried back across the joint and threaded through the scar and into normal tendon. The tension in the graft is adjusted so that the joints are flexed to the same degree as the fingers on each side when the wrist is flexed or extended. The graft is then sutured into place with fine non-absorbable sutures. The lateral slips are brought toward the midline with suture staying distal to the joint so as not to interfere with flexion. It is important to obtain the correct degree of tension over the joint so that the finger may flex and extend properly and the graft will not tear loose later. The injured finger is then splinted in extension for three weeks before movement is allowed.[8]

Rank and Wakefield[10] suggest that in cases in which the central slip of the tendon has been injured close to the insertion, the two lateral slips of expansion be brought into the midline across the back of the joint. In this position they are brought together to prevent recurrence of their lateral slide. Occasionally they suggest dividing each slip or suturing it to the opposite side in a crisscross arrangement. We have used Fowler's[11] method on two occasions with good results. This consists of employing a thin tendon graft (a portion of the palmaris longus or one of the extensor tendons of the toes). A drill hole is placed through the distal third of the middle phalanx. Employing the proximal slips of the interossei as motor

power, one tendon is sutured to the interosseus on one side. It is then brought subcutaneously across the middle finger joint to the opposite side through the drill hole in the bone and then proximally across the joint again to the opposite interosseus tendon.

Dislocation of the extensor tendons over the metacarpophalangeal joints is a rare condition, only a few cases having been reported.[5] Dislocation may be due to trauma and of the "buttonhole" type with the metacarpal head protruding through the tear in the extensor aponeurosis. The most frequent cause is some pathologic change, such as deforming arthritic changes about the joints, or to some central nervous system disturbance associated with paralysis.

Few symptoms are caused; weakness and easy tiring of the finger are noted. During flexion the tendon may be felt to snap down onto the side of the metacarpal head and some difficulty results in extending the finger. Pain and some tenderness are present in the traumatic cases.

In early cases splinting in extension may lead to restoration of function to normal; with older cases, however, operative treatment is necessary. The dislocated tendon must be brought into position and held there by means of fascial bands over the head of the metacarpal and to the neighboring tendon.[5]

Extensor tendons may rupture at the wrist. Trauma is the most frequent cause for so-called spontaneous rupture and the extensor pollicis longus tendon is most frequently involved. Trevor,[12] in 1950, found seventy-six reported cases of subcutaneous rupture of the extensor pollicis longus tendon following Colles' fracture in the literature, and added nine cases of his own. Coburn[13] reported one case in 1945 and Weinberg[14] reported three cases in 1950 which are not included in the aforementioned series. The incidence of rupture of the extensor pollicis longus tendon occurs roughly once in 250 cases of Colles' fracture.[13] Recently Levine and Schneider[15] reported a case of spontaneous rupture of the extensor pollicis longus tendon following a fracture of a carpal navicular bone. In our series of cases there were four patients with ruptured extensor pollicis longus tendons following Colles' fractures. In one case separation occurred spontaneously following minor trauma.

Of the reported cases, 60 per cent are females and 40 per cent are males.[11] The right hand is affected in 80 per cent of the cases.[5] The tendon rupture may occur in from twelve days to seven years, although two to seven months seems to be the average time. The condition can occur at any age but it is more common after the age of thirty. The youngest case reported was that of a fourteen year old girl.[16] The most widely accepted theory as to etiology is interference with the blood supply of the tendon at the time of wrist fracture with aseptic necrosis and eventual rupture of the tendon.

Rupture of the extensor pollicis longus tendon has been described as due to chronic trauma. It was first described in drummers in the German Army.[5] Occupation has been shown to predispose to the condition. It seems more likely to develop in waiters, tailors,[17] furniture polishers, carpenters,[18] rubber workers and wood carvers. Excessive use of the tendon with angulation leads to a chronic tenosynovitis with pain and swelling. The chronic aseptic inflammation continues with persistence of the trauma and leads to eventual division of the fibers.

In addition, chronic tuberculous tenosynovitis may lead to rupture of the extensor pollicis longus. We encountered one case of this type of rupture. The history given by the patient is usually sudden loss of ability to extend the distal phalanx of the thumb, not accompanied by pain. The patient may note a giving way or hear a cracking sound just preceding the loss of tendon function.

On examination the thumb cannot be extended at the distal phalanx and the first metacarpal cannot be elevated to the level of the second. The ulnar border of the anatomic snuff-box is gone due to the separation of the tendon ends.

Operative treatment is the only method of restoration of function; splinting in extension has never led to spontaneous repair. Occasionally it may be possible to suture the tendon ends primarily. Usually, however, approximation of the ends cannot be obtained. The extensor indicis proprius has been used as a transfer to the distal segment of the extensor pollicis with good results. Trevor[12] reported nine cases in 1950 in which he approximated the ends of the tendon as nearly as possible with nylon suture with the thumb in hyperextension. The gap in one case was ¾ inch, usually only ¼ inch. The thumb was then put in a cast in hyperextension, allowing slight mo-

tion of the interphalangeal joint. The cast was removed in three weeks and motion begun. Full mobility of the thumb was regained. In four cases it was later necessary to remove the nylon due to sinus formation. In each case fibrous tissue, similar in thickness to the tendon, had filled the gap. In our experience a tendon graft, from the palmaris longus or from the long extensor tendons of the toes to fill the defect in the extensor pollicis longus, is the procedure of choice, and this method gives the best results. Use of the extensor indicis proprius also gives good results. The extensor carpi radialis does not work as well as these two methods.

TABLE I
RUPTURE OF EXTENSOR TENDONS

Tendon	Etiology	Result
E.P.L.* (thumb).....	Fractured radius	Transplant, extensor indicis proprius
E.P.L.....	Fractured radius	Graft, palmaris longus
E.P.L.....	Fractured radius	No surgery
E.P.L.....	Slight trauma	Graft, palmaris longus
E.P.L.....	Tuberculous tenosynovitis	Transplant, extensor indicus proprius
E.D.C.† (index and middle fingers)	Punch press	Transplant, extensor indicis proprius

* Extensor pollicis longus.
† Extensor digitorum communis.

Two cases of rupture of the extensor tendons at the inferior radioulnar joint are reported by Vaughn-Jackson.[19] One was a fifty-seven year old laborer who painlessly lost the power of extension of the left little finger. One week later, in the same manner, he lost extension power in the left ring finger and noticed a lump toward the ulnar side of the hand.

At operation the tendons of the extensor digitorum communis and the extensor digiti minimi to the ring and little fingers were found to be ruptured, and the extensor digitorum communis to the middle finger was frayed. There was a hole in the joint capsule over the lower articular surface of the ulna and a roughened arthritic area of ulna showed through on pronation and supination of the forearm. This was resected and the tendons were grafted with good return of function.

The second case was exactly similar except there was no involvement of the extensor digitorum communis to the middle finger. It was treated in the same manner.

We had six cases of ruptures of the extensor tendons, at the wrist. Five ruptures were due to trauma, three of which followed fractures of the distal end of the radius; and one was due to tenosynovitis. (Table I.)

FLEXOR TENDONS

Rupture of a flexor tendon is extremely rare, only a few cases having been reported. The ruptured flexor tendon appears to follow a sudden extension of a tightly flexed finger. As a rule the rupture occurs at the tendon insertion on the distal phalanx, but rupture may occur anywhere along the tendon course. Rupture of the flexor digitorum profundus tendon to the index finger and the flexor pollicis tendon at the wrist due to Kienböck's disease has been reported.[20] In addition, rupture of the flexor pollicis longus after Colles' fracture has also been reported.[16] Ruptured flexor tendons may appear following chronic tuberculous tenosynovitis.

Bunnell[21] reports sixteen cases of ruptured flexor tendons from his file and discusses five found in the literature.

In our series we had twelve cases of ruptured flexor tendons. Nine of these were due to trauma and three to tenosynovitis. Improved function was noticed in all patients operated upon. (Table II.)

Usually the patient does not experience any pain at the time of rupture of a flexor tendon and first notices that he cannot flex the injured finger or thumb. Swelling and pain soon follow the injury, however.

Diagnosis is made on history and the inability of the patient to voluntarily flex the distal phalanx which is usually hyperextended due to the unopposed pull of the extensor tendon. Thinning of the distal two phalanges may be noted in the anteroposterior diameter as compared to the normal member of the opposite hand. X-ray examination may reveal a small shell of bone pulled away from the distal phalanx if the rupture occurs at the tendon insertion.

When a ruptured tendon is seen late, tendon grafting will probably be necessary since there will be apparent loss of substance due to muscle and tendon contracture. The palmaris longus tendon and the long extensor tendons to the second and third toes are satisfactory for grafting. Paratenon is important, for without it the graft will adhere to the surrounding tissue with

resulting limitation of motion. For this reason it is wise to make an incision the length of the desired graft along the course of the tendon to be taken.

Following operation for tendon repair or tendon graft, the hand should be splinted in a position to relax the repaired tendon: moderate extension for the extensor tendons and moderate flexion for flexor tendons. The fingers are only moderately flexed. The relaxation is obtained at the wrist. The splint should extend well up on the forearm and a compression type of dressing applied. The part is kept immobilized for three weeks. During the fourth week purposeful motion is allowed but the splint is retained except for periods of exercise. After the fourth week voluntary motion is encouraged and the splint is discarded. For extensor tendons the splint is usually left on for four weeks.

TABLE II
RUPTURE OF FLEXOR TENDONS

Tendon	Etiology	Result
F.D.P.* (ring finger)	Football	Tendon graft; good result
F.D.P. (middle finger)	Pulling bar	No surgery
F.P.L.† (thumb)	Tenosynovitis	Tendon graft; good result
F.D.P. (little finger)	Basketball	Arthrodesis of distal joints
F.P.L. (thumb)	Shovel	No surgery
F.P.L. (thumb)	Baseball	No surgery
F.D.P. (ring and middle finger)	Punch press	Surgery; amputation
F.D.P. (little and ring fingers)	Tuberculosis	Surgery; limitation of motion
F.D.P. (index finger)	Football	Surgery; good motion
F.D.S.‡ (little finger)	Tuberculosis	Good; recurrence of tuberculosis
F.P.L. (thumb) F.D.P. (index finger) F.D.S. (index finger)	Fourteen year old fracture of radius and ulna	Surgery not followed long enough
All tendons and nerve at wrist	Homemade bomb	Skin graft; definitive surgery not done

* Flexor digitorum profundus.
† Flexor pollicis longus.
‡ Flexor digitorum sublimis.

CASE REPORTS

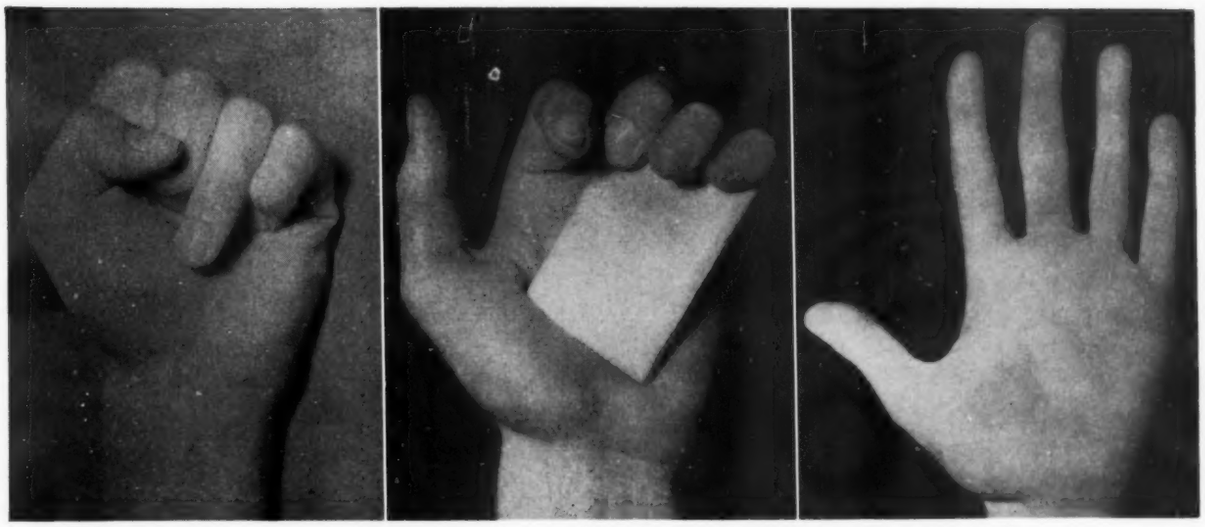
CASE I. This seventeen year old boy first noted his injury while playing football eleven months prior to being seen by us. During a huddle he noticed the left ring finger did not grip the knee as the other fingers did. He was unable to flex the tip of the finger. There was no pain and he remembered no injury. He was taken out of the game and only then did he notice pain. The finger became moderately swollen. He was seen by a number of physicians before it was recognized that he had a rupture of the flexor digitorum profundus tendon of the left ring finger. Examination eleven months after injury revealed that the middle and distal phalanges of the left ring finger were somewhat thinner in anteroposterior diameter than the normal phalanges on the right. The patient was unable to voluntarily flex the distal phalanx of the left ring finger. Extension of the distal phalanx was good; it was held in a position of slight hyperextension. Passive flexion was normal; the patient was able to touch the palm. There was no sensory loss. Motions of all other joints were normal.

At operation the flexor profundus tendon was found avulsed from the distal phalanx and its proximal portion was adherent to the flexor digitorum sublimis in the distal portion of the proximal phalanx. This was freed up and brought back out into the palm.

The long extensor tendon to the third left toe was taken for a graft and was united to the base of the distal phalanx, after exposing raw bone, with a pull-out wire suture tied over a button on the nail surface. The graft was threaded proximally, going around the sublimis insertion and into the palm where it was attached to the proximal portion of the flexor profundus tendon by threading through a slit in the tendon and suturing in position with No. 60 cotton. The patient now has strength in the distal phalanx and motion, but is unable to bring the tip well down into the palm on flexion. It will probably be necessary to shorten this tendon at a second operation. (Fig. 1.)

CASE II. This forty-six year old oilfield worker suffered an injury to his right long finger in September, 1950. He stated that he was pulling on a pry bar while working on a drill rod when he suddenly felt something give way in his finger. Subsequently, exploration of the long finger was carried out elsewhere. At operation the detached fragment of bone was found lying in the joint space anteriorly. No flexor digitorum profundus was found. A tendon transplant was not considered feasible because of the rather marked atrophy in the region of the finger.

In March, 1951, Dupuytren's contracture of the right hand was corrected. At that time the profundus to the long finger was removed and tendolysis was performed on the flexor digitorum sublimis. Following this last procedure it was noted that there was a fair range of motion. In January, 1954, he complained that the long finger was stiff



1A 1B 1C

FIG. 1. Case 1. Patient was a seventeen year old high school student. A, rupture of the flexor digitorum profundus to the left ring finger. B, flexor power noted following a tendon graft. C, extension.

and had a tendency to get in the way. In addition, signs of causalgia, coldness, dampness, atrophy and the like developed in the entire right hand. He was given a cervical sympathetic block with relief.

When first seen by us on February 1, 1954, he complained of inability to flex his right middle finger. Active flexion was possible only to within 2¾ inches of the palm. Atrophy of the finger was noted. X-ray examination taken on October 7, 1950, had revealed an avulsion of a fragment of bone from the proximal portion of the distal phalanx of the right middle finger. (Fig. 2.)

Further reconstructive procedures, such as a tendon graft, were considered but the patient decided that he did not want any further surgery performed.

CASE III.* O. K. K., was first seen on February 29, 1952, with a history of injury to his right thumb about five days previously. He stated that while tightening a wing nut on a clamp he felt something move in his thumb, after which he was unable to bend it. Examination revealed inability to flex the thumb. Sensation was intact and good passive motion of the joints was present.

The patient was admitted to the hospital and the operative report is as follows:

"A mid-lateral incision was made on the lateral aspect of the thumb and the flexor pollicis longus tendon was identified. It was found to have narrowed and to have ruptured about the mid-portion of the thenar eminence. The distal portion was brought out through the incision in the thumb itself.

* This case was originally presented in: POSCH, J. L. *Am. J. Surg.*, 85: 306–318, 1953.[22]

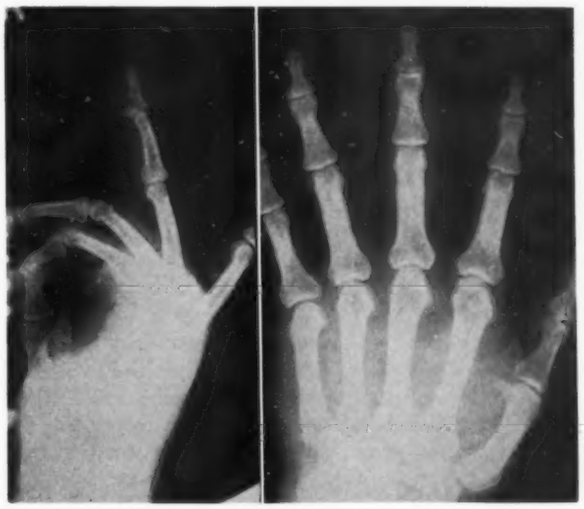
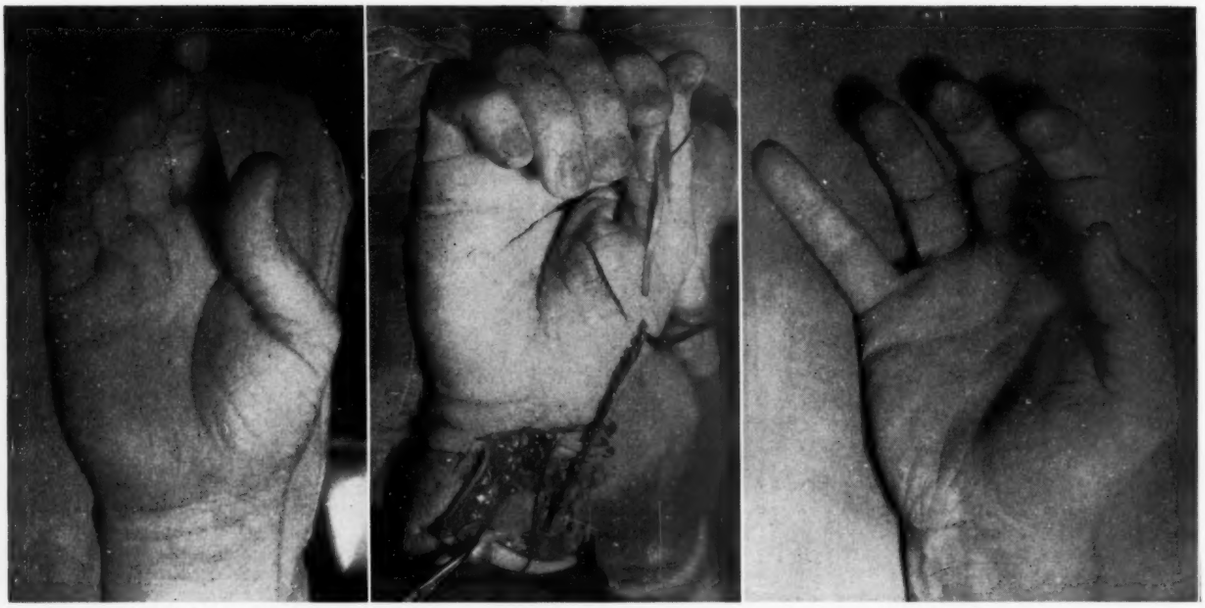


FIG. 2. Case II. Roentgen ray of avulsion of flexor digitorum profundus from distal phalanx of right long finger.

"A transverse incision was then made in the wrist and the flexor pollicis longus was identified here. This was done with some difficulty because the flexor pollicis longus tendon had become quite adherent and it was difficult to distinguish it from the flexor digitorum profundus to the index finger. However, by enlarging the incision proximalward along the radial side of the wrist for about 7 cm. the origin of the flexor pollicis longus was identified.

"A small transverse incision was then made at about the mid portion of the thenar eminence and the flexor tendon sheath of the flexor pollicis longus was identified here. By means of tendon strippers



3A 3B 3C

FIG. 3. Case III. A, rupture of flexor pollicis longus following minor trauma. B, findings at surgery. C, flexor power six months following tendon grafting procedures. (From POSCH, J. L. *Am. J. Surg.*, 85: 306–318, 1953.[22])

the proximal portion of the flexor pollicis longus tendon was freed from scar tissue and brought out in the wrist. It was noted that through some diseased process the flexor pollicis longus tendon had gradually atrophied and eventually ruptured at about the midportion of its course over the midportion of the thenar eminence. Since it was thought that even with suturing the tendon would not be strong enough for a good result, it was decided to do a tendon graft. Accordingly, the long extensor of the third toe of the right foot was removed for approximately seven inches and then sutured to the distal portion of the flexor pollicis longus tendon in the thenar eminence and to the proximal portion of the tendon in the wrist. This was accomplished first by suturing it on the wrist and then tunneling it by means of tendon leaders into the thenar eminence. Here it was then also approximated very carefully end to end. This was done with interrupted No. 60 cotton sutures."

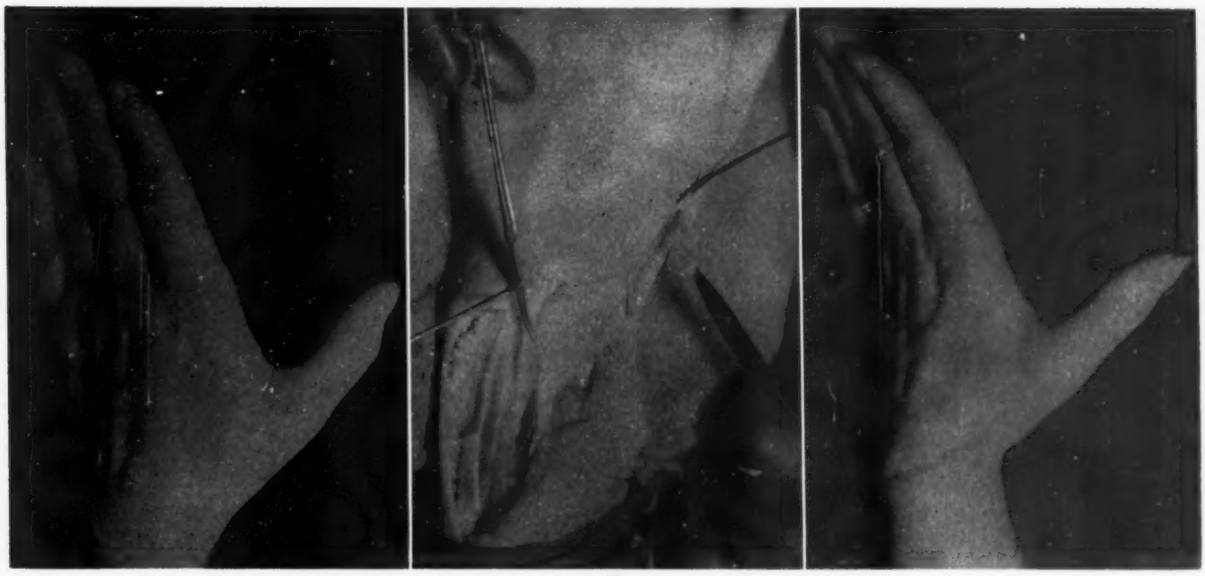
The microscopic diagnosis of the ruptured portion of the tendon that was sent to the laboratory was chronic synovitis of tendon.

The patient's postoperative course was uneventful and he was discharged from the hospital one week postoperatively. The posterior splint was removed at the end of four weeks, and at first motion was very slow. However, he gradually received strength in the end of his thumb. When last seen he was able to use it for writing and in all of his daily work. (Fig. 3.)

CASE IV. J. W., a fifty-two year old white woman, fell at work and suffered a Colles' fracture of the left wrist. The fracture was reduced and the patient had an uneventful course. When the cast was removed it was found she could no longer extend the tip of the thumb. At operation the extensor pollicis longus tendon was found to be ruptured at the radial styloid. The extensor indicis proprius was sectioned at the metacarpophalangeal joint and used as a tendon transplant to the distal segment of the extensor pollicis longus. The patient had good return of function. (Fig. 4.)

CASE V. T. R., a fourteen year old boy, was struck on the tip of the partially flexed left index finger while grasping for a fast moving football on October 26, 1953. The force of the blow caused a sudden hyperextension of this finger resulting in pain and tenderness over the flexor surface of the distal interphalangeal joint. A roentgenogram taken showed a small fragment fracture of the base of the distal phalanx. A splint was applied with the finger in slight flexion and the patient was referred to the Allentown Hand Clinic. Two days after the initial injury an examination disclosed complete loss of active flexion of the distal interphalangeal joint of the left index finger. A bulbous thickening at the distal interphalangeal joint acted as a block to complete passive flexion of this joint. There was no limitation of extension and sensation was normal.

One week after injury surgery was performed at the Allentown Hospital. Exposure of the terminal interphalangeal joint disclosed the flexor profundus tendon to be completely torn loose from its insertion into the distal phalanx. A spicule of bone had been carried downward with the avulsed tendon.



4A 4B 4C

FIG. 4. Case IV. A, rupture of extensor pollicis longus following a fracture of the distal end of the radius. B, findings at surgery; note the widely separated tendon end held in forceps. C, extension possible four months following tendon transplant.

Moderate early periarticular fibrosis was encountered. The severed end was firmly adherent to the joint capsule and radial digital nerve. Filmy adhesions extended into the tendon sheath to the point of insertion of the sublimis tendon. The profundus tendon was released from its adherent bed, the edges freshened and the stump fixed to the bone after the technic of Bunnell, applying a pull-out wire passed through a small drill hole in the bone. The skin and subcutaneous tissues were carefully approximated and the finger and wrist splinted in flexion with maximum flexion at the metacarpophalangeal and wrist joints. Three weeks postoperatively the wire was removed and active motion continued. Prior to removal of this wire, biweekly active exercises in flexion were given in the office. Complete range of flexion was obtained forty-one days after repair. (Fig. 5.) A 5 degree flexion deformity of the index finger was noted when the latter was fully extended. One year after repair the patient presented a normally extended finger with good grasp and excellent flexion function.

CASE VI. M. L., a twenty-eight year old woman, reported to the Hand Clinic at the Allentown Hospital because of inability to completely flex the fifth finger of the right hand. She was not particularly concerned about the cystic swelling over her right wrist. Gradual onset of wrist swelling occurred about two years preoperatively. Flexion deformity of the fifth finger occurred about one year preoperatively and more recently had been accompanied by pain which radiated up the ulnar side of the forearm. With the finger held in extension at the proximal interphalangeal joint, the metacarpophalangeal joint flexion of the terminal phalanx was 90 per cent of normal. There was loss of ability to flex the proximal interphalangeal joint of this finger. Except for this flexion contracture there was no motion disability. No sensory loss was noted.

The tuberculous process was found to have involved the radial and ulnar bursae and to have spread into the mid-palmar space. The flexor sublimis tendons of the fifth finger were fragmented and shreaded and it was necessary to completely excise this tendon together with the mass of tuberculous tissue in the wrist. The carpal tunnel was completely cleaned out. The dissection included both parietal and visceral layers of the tendon sheaths.

Except for several small areas of aseptic necrosis of the skin flaps, the patient made an uneventful recovery with return of function of the fifth finger.

Recent examination, however, revealed a thickening along the course of the flexor tendons to the index finger. I believe that there is a recurrence of the tuberculous process and re-exploration is indicated at present. (Fig. 6.)

CASE VII. M. S., a fifty-four year old man employed as a cutter in a textile factory, reported to the Hand Clinic at the Allentown Hospital complaining of inability to extend his right thumb. This disability had occurred twenty-four hours prior to admission to the clinic. While cleaning his roof he picked up a stick which he used to shoo

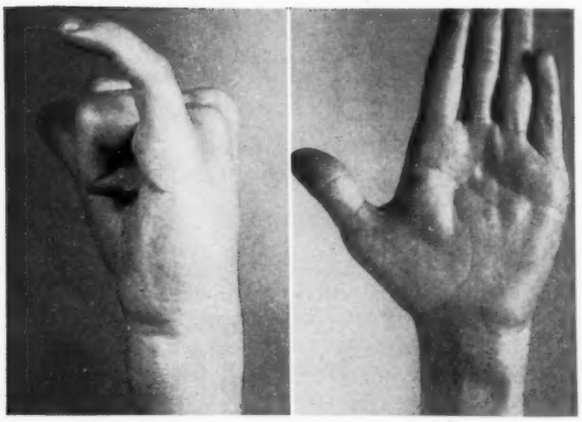
5A 5B 5C 5D

FIG. 5. Case v. A, findings at surgery of a ruptured flexor digitorum profundus to the left index finger following injury by a football. B, suturing of flexor digitorum profundus at distal phalanx. C, flexion possible forty-one days following repair. D, extension.

away pigeons perched on the chimney. During this act he suddenly felt a snap in the region of the anatomic snuffbox. There was an immediate loss of the power to extend the distal joint of the thumb. Dorsiflexion and radial abduction of the wrist joint were not impaired. There was no loss of sensation in the thumb.

There was no previous history of fracture to the wrist bones nor was there any x-ray evidence of underlying disease in the carpus or metacarpus.

One year prior to the present episode the patient had had a severe coronary attack and was hospitalized for three months. Right hand-shoulder syndrome noted at this time resulted in edema and



6A 6B

FIG. 6A and B. Case VI. A, flexion deformity of the right little finger due to destruction of the flexor digitorium sublimis by tuberculous tensynovitis. B, note cystic swelling along the course of the radial and ulnar bursae.

6C 6D

FIG. 6C and D. Same case as Figure 6A and B. Findings at surgery.

swelling of the hand with some stiffness which was relieved by physiotherapy and resulted in complete return of function.

Repair of the fragmented thickened tendon was accomplished by excision of the extensor pollicis longus and insertion of a palmaris longus graft. The patient made an uneventful recovery and had complete return of function.

CASE VIII. H. E., a thirty-three year old white woman, had always been in good health. Fourteen years previous to admission she sustained a fracture of the right radius and ulna. Healing was prompt and functional return complete.

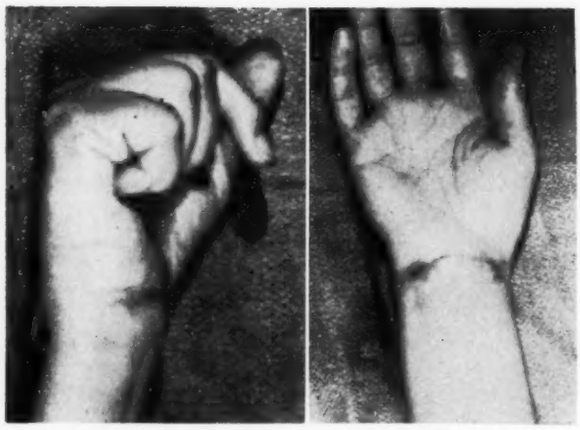
Four years prior to admission, while at work, she suddenly noted inability to flex the terminal phalanx of the right thumb. At this time she thought that arthritis was developing; x-rays demonstrated an old healed fracture of the radius. The loss of flexion function of the thumb was

FIG. 6E. Same case as Figures 6A to D. Postoperative photograph showing amount of extension possible; flexion also good.

sudden and not accompanied by pain. There was no anesthesia of the thumb nor was there any tenderness experienced along the course of the flexor pollicis longus.

Two years before admission, while continuing her work as a "sleever," she noted that she was unable to flex the terminal phalanx of the right index finger. Her work required that she have a firm pinch in order to approximate the polo shirt sleeve to its body. The loss of terminal phalangeal flexion resulted in some clumsiness and decreased her work efficiency. She was able, however, to adjust to the loss of terminal phalangeal flexion by firm flexion of the proximal interphalangeal joint. However, five months previously, while engaged as a "sleever," she experienced a snapping painful sensation beginning in her wrist and radiating down the thenar eminence and up into the forearm. At this time she noted loss of flexion at the proximal interphalangeal joint of the index finger. With the inability to perform pinch, she was forced to leave her job.

Exploration of the wrist was done at the Allentown Hospital on February 1, 1955. The surgeon described the findings as "a sloughing away of the flexor pollicis longus tendon and the profundus and



7A 7B

FIG. 7. A, rupture of flexor pollicis longus and flexor digitorum profundus and sublimis to the index finger following an old fracture of the right radius and ulna; flexion possible following a tendon graft to the index finger. B, extension.

sublimis tendons to the index finger at the wrist joint." Exploration was performed under local anesthesia and definitive surgery was discontinued after lateral suture of the distal stumps of the index flexor tendon to the flexor tendons of the long finger. A cast was applied for three weeks and the patient received active physiotherapy for another three weeks. She did obtain 30 degrees of flexion at the proximal interphalangeal joint and 10 degrees at the distal interphalangeal joint. No attempts were made at this operation to repair the pollicis longus tendon.

In view of continued disability reoperation was performed on May 4, 1955, revealing complete disruption of the flexor pollicis longus and superficial and deep flexor tendons to the index finger of the right hand. Fragmentation of the tendons to the index finger was marked. There was no connection between the muscle belly and the long flexor tendon of the thumb. A gap of approximately 4 inches existed. The muscle belly of the flexor of the thumb was atrophied and of little use as a motor. The muscle masses of the profundus and the sublimis tendon to the index finger were contracted and fibrotic but reacted well on stimulation. A palmaris longus graft was fixed to the stumps of the profundus and sublimis tendons in the palm, drawn up in the wrist and fixed to their muscle bellies. The flexor sublimis of the ring finger was utilized to obtain flexion of the terminal phalanx of the thumb. The postoperative course was uneventful. The patient is still in the convalescent stage. (Figs. 7A and B.)

CONCLUSION

A series of ruptured tendons of the hand and wrist have been presented. The historical aspects and the etiologic factors have been emphasized. Attempts were made to illustrate the diagnostic difficulties often encountered in this type of injury. We hope that in the future they may be more readily recognized.

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Tendolysis within the Digit Using Hydrocortisone Locally and Early Postoperative Motion

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THE adherence of tendon to scar tissue within a digit constitutes one of the most difficult problems with which the surgeon must deal in surgery of the hand. The adherence of tendon to periosteum, tendon tunnel or ligament acts in effect as a new insertion of the tendon in that area, preventing it from transmitting force to more distal joints. The greater difficulty of successful tendon repair within the digit, as compared to the forearm or palm, is well recognized for these reasons. In view of this there is a growing tendency not to repair tendons within a digit, but rather to carry out a delayed tendon graft.[1,2] If scarring is severe, replacement of soft tissues by pedicle graft may be necessary before the tendon graft is carried out.

The reported effects of cortisone and its derivatives on the healing of tendon and on the prevention of adhesions about tendons in the experimental animal have been variable. This is due, most likely, to different experimental conditions, variations in dosage and variable effects in different species of animals. Grant[3] found no interference with healing of the Achilles tendon in rabbits with a daily dose of 7.5 mg./kg. of cortisone when the tendons were divided halfway across and resutured. Carstam[4] observed significant depression of adhesion formation about scarified tendons in the rabbit with a daily dose of 2 to 4 mg./kg. of cortisone and a tendency of completely divided and resutured tendons to separate if they were not immobilized in relaxation. Gonzales[5] found repaired flexor tendons in the forepaw of the dog no more free and of essentially the same tensile strength as the controls after three weeks' immobilization in casts using 0.25 cc. hydrocortisone locally in the surgical wound. Baxter et al.[6] observed clinical benefit from the systemic administration of cortisone following operation for Dupuytren's contracture.

Following the demonstration of the effectiveness of the local use of hydrocortisone in joints by Hollander et al.,[7] it was used locally in the hand in a variety of clinical conditions by Howard, Pratt and Bunnell.[8] In six patients the material was used locally in the wound following tendon freeing with good results. A single complication occurred in a digit in which osteotomy of the proximal phalanx was performed at the same time. The tendon remained quite free, but bony union was not complete at the end of three months. The principle advantage of the hydrocortisone seemed to be the fact that there was minimal postoperative pain and reaction and this allowed earlier exercise of the freed tendon. Zachariae and Zachariae[9] have reported favorably on the local use of hydrocortisone in the treatment of post-traumatic stiffness of the hand.

The anti-inflammatory qualities of hydrocortisone have seemed particularly desirable following tendon-freeing operations in order to encourage early motion postoperatively. With that in mind, we have used the material locally at the time of operation in sixteen patients and have tried to evaluate its effect.*

The patients in the cases reported herein were operated upon to free adhesions about tendons within the digit following crush injury (four cases), tendon repair (nine cases) or grafting (three cases). Three cases involved extensor tendon, twelve involved flexor tendon and one case involved both flexor and extensor tendon in the same digit, a most difficult situation. Operation ideally should not be undertaken

* The material used in this study was hydrocortone,® 25 mg. per ml., kindly supplied by Merck & Co., Rahway, New Jersey.

unless the joints are freely mobile. Any joint disability has an adverse effect upon results.

The digits were entered through longitudinal mid-lateral incisions which gave excellent exposure of either the flexor or extensor mechanisms by elevation of the appropriate flap.

Peripheral block anesthesia was used in most instances. The ulnar nerve was blocked at the elbow and the median and radial nerves were blocked at the wrist. All procedures were carried out in a bloodless field with a pneumatic cuff tourniquet in place. Skin preparation involved mechanical cleansing with phisoderm® and saline the night before and immediately preceding surgery. The tendons were completely freed by sharp dissection so that tension applied to the tendon, through a complimentary incision, either in the palm or dorsum of the hand, gave a complete range of motion. Needless to say, this is an essential part of the procedure. If adhesions to the tendon are left, the procedure is doomed to failure before the operation is completed. An attempt is made to preserve at least some of the tendon tunnels to prevent bowing of the tendon postoperatively.

Once the tendon is free, the great problem is to keep it that way. A thin layer of fascia from the extensor muscles of the forearm was employed on one side of the tendon in five cases. At the close of the operation 1 to 2 cc. (25 to 50 mg.) of hydrocortisone was distributed along the tendon. In order to keep it within the finger this has been injected through a small polyethylene catheter after the finger is closed. The catheter is left alongside the tendon emerging from the proximal wound. After the injection, it is removed to reduce irritation and diminish the chance of infection. No subsequent injections of hydrocortisone have been used in this series of cases.

The most important factor in preventing the reformation of adhesions about tendons is early and complete active motion postoperatively. A very loose soft dressing with kerlix® gauze is used to interfere as little as possible with motion. It has been our impression that the patients have moved their digits with less pain when hydrocortisone is employed at operation.

The patients are kept in the hospital for four or five days during which time they are encouraged to move their digits actively. An antibiotic is administered and in some instances, if active motion is not good, peripheral nerve blocks at the wrist are employed using 2 per cent xylocaine. The blocks are an aid to motion not only in their actual benefit but also as a psychologic stimulus. After a patient has had a peripheral block he will usually work harder to avoid a subsequent one. Another stimulus to active motion is the time of discharge from the hospital. When the patient asks, "When may I go home?", the reply is, "When you are moving your finger well." Usually the time of discharge is the fourth or fifth postoperative day.

COMMENTS

Table 1 is a summary of the sixteen cases included in this series. In three of these more than one finger was involved, making a total of nineteen digits. It is interesting that all but two of the cases involved the right hand. There were six cases involving the index finger, six cases involving the long finger, three cases involving the ring finger, three cases involving the small finger and one case involving the thumb. The time between the original injury and tendolysis varied from four months to seven years and four months. A layer of fascia was employed on one side of the tendon in five of the fingers. The dosage of hydrocortisone employed varied from 25 to 50 mg. applied locally at the close of the operation. No subsequent injections were made in this series. The higher dosages have been used in the more recent cases.

Table 11 is a summary of results. It is seen that good to excellent results were obtained in approximately half of the cases and fair results in an additional 32 per cent. In short, worth while results were obtained in 84 per cent of the digits operated upon.

The two cases (1 and 10) which were considered to have poor results each had very severe associated deformities. The patient in Case 1 had adherence of the extensor as well as the flexor tendon due to a buzz saw injury which also had injured the proximal joint of the index finger. In our experience adherence of both flexor and extensor tendons in the same digit is most difficult to cure. It can be seen that some improvement was obtained in this case, but it is classified as poor on the basis of a persistent 70 degree lack of extension of the middle joint. The patient, however, is satisfied. The patient in Case 10 had associated joint stiffness and a low pain threshold which were

TABLE 1
SUMMARY OF CASES

Case No.	Date of Injury	Digit	Tendon	Operation	Fascia	Hydrocortisone (mg.)	Degrees*		Cm.†		Result
							Preoperative	Postoperative	Preoperative	Postoperative	
1	5/7/52	Left index	Extensor and flexor	3/12/53	0	0	82	45	10	1	.....
	5/7/52	Left index	Extensor and flexor	9/8/53	0	25	45	70	1	0	Poor
2	4/14/51	Right ring	Flexor	4/17/53	0	25	0	0	7.5	1.5	Good
		Right small	Flexor	4/17/53	0	25	0	0	7	0	Excellent
3	1/–/46	Right long	Flexor	4/22/53	0	25	40	30	0	0	Fair
4	1/4/53	Right long	Extensor	5/1/53	X	25	54	32	6	0	Fair
5	2/–/46	Right long	Flexor	6/10/53	0	12.5	0	8	4.5	0	Excellent
6	8/21/52	Right small	Flexor	12/15/52	X	0	0	0	4.5	4.5	.....
	8/21/52	Right small	Flexor	10/5/53	0	25	0	5	4.5	1	Good
7	3/3/53	Left ring	Flexor	10/10/53	0	25	0	0	2.5	1	Fair
8	8/30/53	Right index	Extensor	11/25/53	X	25	28	30	5.5	3	Fair
9	8/28/53	Right small	Flexor	1/27/54	0	25	8	0	3	0	Excellent
10	4/28/52	Right index	Flexor	2/15/54	0	12.5	42	40	4	3.5	Poor
		Right thumb	Flexor	2/15/54	0	12.5	..	..	4	4	Poor
11	8/24/53	Right index	Flexor	4/1/54	0	40	55	45	4	2.5	Fair
12	10/3/53	Right long	Flexor	7/12/54	X	50	40	5	2.2	0.5	Excellent
13	7/24/54	Right long	Flexor	11/18/54	0	50	70	4	2.5	0	Excellent
14	4/26/54	Right long	Flexor	1/7/55	0	25	0	0	2	0	Excellent
	4/26/54	Right ring	Flexor	1/7/55	0	25	3	0	1.8	0.4	Excellent
15	4/27/54	Right index	Flexor	5/9/55	0	40	0	5	9	3	Good
16	8/26/53	Right index	Extensor	5/11/55	X	40	0	0	5.5	2	Fair

* Degrees: Lack of complete extension of middle joint.
† Cm.: Lack of flexion to palm at base of finger.

factors in the poor result obtained. She does have useful grasp, however, between the thumb and index finger.

The patient in Case 6 is a physician upon whom two operations were performed for adherence of the flexor tendons of the right small finger. At the first operation a fascial graft was used on one side of the tendon and postoperative wrist blocks were employed without success. At the second tendolysis ten months later, employing hydrocortisone and no fascia, a good result was obtained. The patient was impressed with the lack of postoperative pain in comparison to the first operation, and moved

his finger much more readily than without the hydrocortisone.

SUMMARY

Nineteen digits in sixteen patients were operated upon to free adherent tendons within the digits. Hydrocortisone was placed locally about the tendons at the close of operation and early motion was carried out postoperatively. Good to excellent results were obtained in 52 per cent and some improvement in an additional 32 per cent.

TABLE II
RESULTS

Tendon	No. of Patients Operated	No. of Digits Operated	Excellent (37%)	Good (15%)	Fair (32%)	Poor (16%)
Flexors.....	12	15	7	3	3	2
Extensors.....	3	3	..	..	3	..
Flexor and extensor.....	1	1	..	..	..	1

CONCLUSION

Tendolysis within the digit has been a worth while operation.

Hydrocortisone, applied locally in the wound at the time of surgery, seems to be a useful adjunct in reducing pain and allowing earlier and more complete postoperative motion.

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DISCUSSION OF PAPERS BY DRS. WHITE, POSCH, WALKER AND MILLER, AND WECKESSER

SUMNER L. KOCH (Chicago, Ill.): I am sure that many of the results obtained by these authors have been better than excellent, and they are to be congratulated. I am sure we all appreciate the objective attitude that they have taken toward their results.

I was interested in a few things that Dr. White mentioned, and I would like to question him about them just briefly. He said, if I understood correctly, that he did not approve of using tendon grafts in children under the age of five, and I wonder why he took that attitude. We have not hesitated in trying to repair divided tendons in children, regardless of age. We are concerned about the atrophy both of the finger and the muscle that seemed to persist if those fingers were not put to work.

I hope that within the near future we will be able to report a series of cases of division of flexor tendons in children—some repaired with sutures, some repaired with grafts—in which we really have been pleased with the results obtained.

One factor, I am sure, is that children provide their own physical therapy because no compensation element is involved. You simply cannot keep them restricted, and I wonder why Dr. White has set that particular age. Perhaps I did not understand his statement correctly, that he thought the primary treatment was not a factor in the results he had obtained. Is that right, Dr. White? (Dr. White answered: In the evaluation of these seventy-six cases it did not appear too significant.)

In our experience this is not so. The primary treatment has often almost defeated us in our attempts to carry out the secondary repair because if thick suture material has resulted in fixation of the tendon to everything around it, it is often exceedingly difficult to excise that fibrous tendon, the thick suture material and so forth, and then obtain a freed lining tendon as a result of tendon transplantation.

We are always happy if patients come to us with simple closure of the wound and without any primary treatment because then we have had the most favorable conditions for getting a good result.

I had hoped that Dr. White would say a little

more about how the tendon graft is attached to the distal phalanx. Perhaps in his discussion he did not have time to mention what he would do if the profundus was divided in the middle phalanx and the sublimis was left intact. As you know, Dr. Gothenberg and others have advocated getting a fixation in such cases, an ankylosis at the distal joint, and being content with that.

We believe that is rather an admission of defeat. We use a graft when the profundus alone has been divided and believe that the results are increasingly good.

I was also interested in the statement that most tendons rupture at their insertion, as reported by Drs. Posch, Walker and Miller. Again, this has not been our experience; rather, the extensor tendon ruptures usually just proximal to the interphalangeal joint, and rarely from a blow on the finger. Almost always it occurs as a result of baseball finger injury, a blow on the tip. Perhaps that was what was meant, a blow on the tip of the finger. I was thinking of a blow on the finger itself.

It is the sudden blow on the tip of the finger, with a finger extended, that causes the baseball finger injury or the rupture of the tendon. In our experience the flexors usually rupture either as a result of a tuberculous tenosynovitis, as described in the paper by Drs. Posch et al., with gradual erosion of the tendon and finally rupture at the musculotendinous junction.

I am sure you have all seen, as we have, the patient who has caught his finger in a door and on trying to drag out his hand, carried away the distal phalanx and a long length of tendon with it, the tendons having ruptured at the musculotendinous junction in the forearm. Recently a patient came to us with such an injury. He had the distal phalanx and a long length of tendons in a paper sack and asked us to put them back.

I am very interested in Dr. Weckesser's results. We have not used that method in any of our operating procedures. I am sure we will continue to watch his results with interest, and hope he will continue his efforts.

WILLIAM H. FRACKELTON (Milwaukee, Wis.): Dr. White restated very well the important principles of tendon grafting and has set forth an objective measure and method for evaluation. Dr. Koch has already commented on the question of grafting in children, and I certainly would wonder if many of the rest of us would not agree with Dr. Koch. I have had only a small series of fourteen patients under five years of age. The results of tendon grafting have been worth while. Perhaps some of us believe that one of the most important points of Dr. White's paper was that of a method of evaluating preoperatively what one might expect from a tendon graft. In other words, the difference in result anticipated, a supple hand versus a congealed hand might well be presented to the patient and to those interested in the patient's welfare. This would tend to preserve the status and value of tendon grafting surgery within the surgical community in which it is done.

The paper by Drs. Miller, Posch and Walker splendidly restates the problem of tendon rupture. The difference between tendons in the hand and forearm which do not rupture within their length, as compared to the Achilles tendon which can rupture within its body, is one of histology.

I was interested in their mention of the problem of dislocation of extensor tendons at the metacarpal phalangeal joint with rupture, because that is a problem not often recognized. By contusion and blow on the dorsum there can be disintegration and then rupture, if you will, of the triangular expansion and interosseous muscle tendon. Dislocation of the finger sideways and weakening of power result.

In addition to the repair which these authors advocated, one can sometimes advantageously transfer the power of the interosseous tendon by supplementary graft or by changing the direction of pull of the extensor indices, for example.

These authors also presented the thought that operation for ruptured tendon was preferable in their hands to that of splinting. Perhaps many of you believe, however, that extensor tendon rupture at the distal joint could be handled quite well in the closed injury by extensor splinting. Splinting is uncomfortable for the patient, but so is an operation. With the finger in hyperextension at the distal joint, flexed at the mid-joint, maintained by a padded gutter metal splint and supervised for a four-week period, results have been good without operation.

WILLIAM L. WHITE (closing): In answer to Dr. Koch's question concerning the age at which tendon grafts should be done, in this series there were three patients under the age of five. In two, a fair end result was obtained; the third patient had a good end result. In suggesting that tendon grafting be delayed until after the age of five, I suppose I have been motivated a bit by the fact that some of the best results obtained were in the individuals who had suffered injury early in life and came in with supple fingers at the age of eight or nine. It has become my opinion that it is better to wait until the tendons are bigger and easier to repair, and the patient has reached the age of cooperation.

I quite agree with Dr. Koch that if it is possible to get the desired end results early, then perhaps grafting should be done then.

As to the primary treatment, my report evaluated a particular series of seventy-six tendon grafts. Statistics do not reflect one's opinion in all cases. I quite agree that primary treatment is very im-

portant. However, in this particular series the results were only slightly better when only skin closure had been done as primary treatment. In thirty-one cases in which tendon anastomosis was attempted primarily at the site of injury, fifteen resulted in excellent to good end results which is about the same distribution as the over-all experience. I did not believe the difference was significant. The attachment at the distal phalanx of the tendon grafts, was always by pull-out suture. The proximal anastomosis was done by silk suture.

I was a bit hesitant to make any issue of the fact that in this series there were five individuals, who had lacerated only the profundus tendon, in whom I had left the sublimis and placed a small tendon graft in the finger to restore the profundus. I must admit that three of these grafts gave the most pleasing results in the series. I believe this is justified in carefully selected cases.

PRENTICE J. WALKER (closing): In answer to Dr. Koch's question about the rupture of the extensor tendon distal phalanx, we meant a blow to the tip of the actively extended finger. We have counted six cases of extensor tendon rupture at the wrist. Five of these were due to trauma: three followed fractures of the distal end of the radius, one was due to frequently repeated minor trauma and one was due to severe trauma.

There was the one case of tenosynovitis. We had also encountered twelve cases of flexor tendon rupture, nine due to trauma and three to tenosynovitis.

This series of rupture tendons of the hand and wrist has been presented with some of the historical review and etiologic factors, and we have tried to emphasize that attempts were made to illustrate the diagnostic features.

We hope that in the future these injuries may be more readily recognized and earlier treatment given.

GEORGE B. SANDERS (Louisville, Ky.): At the outset, may I say partly in jest and certainly not in a critical sense, that I think Dr. Weckesser is expecting a lot of hard work from the 1 or 2 cc. of hydrocortisone that he instills into the operative site at the time of operation. In our experience, the effect of single doses of injected hydrocortisone is limited and perhaps lasts little longer than forty-eight to seventy-two hours. We have preferred, in our practice, to employ cortisone or meticorten fairly liberally in difficult hand cases in which we expect much swelling, stiffness and perhaps poor cooperation. We believe, as you all do, that the corticoids do not cure anything, but do limit tissue reaction markedly so as to permit freer use of the part in active and passive exercise, and thereby perhaps get the patient over a critical hump in his rehabilitation from the third to the eighth week postoperatively. It has been our sad experience at times to note the blossoming of symptoms of swelling, stiffness and pain as soon as cortisone has been withdrawn. In several instances we have repeated the course of cortisone once, twice and even three times in an effort to continue patient cooperation in exercise, fairly free of pain, until such time as good cooperation is permanently assured and excessive reaction is a thing of the past. In general, we employ cortisone in three ways. First, preoperatively, in patients who have had previous operations or injuries to the hand and fingers and who are still in the stage of excessive tissue reaction. We believe we can avoid a prolonged waiting period, amounting to many months at times, during which the tissues are hyper-reactive and will not tolerate further operative intervention. Cortisone, started preoperatively, is continued throughout the operative and postoperative period. Second, we have given it postoperatively from the fourth to the eighth week, usually, if tissue hyper-reactivity develops unexpectedly in this period. In such instances we will continue cortisone until its withdrawal no longer produces marked hyper-reactivity of tissue with pain, stiffness and the like. We delay its administration until the fourth week in cases of tendon repair and other similar entities, so as to avoid the undoubted suppression of fibroplasia which the corticoids produce. Third, we employ cortisone somewhat routinely beginning three and a half weeks postoperatively in individuals whom we consider highly susceptible to the development of extreme tissue reactivity: the type of patient who will develop a frozen hand, a shoulder-hand syndrome or similar neurocirculatory phenomenon. In these instances we do not wait for the signs of hyper-reactivity to appear and we routinely begin cortisone administration at three and one-half weeks when active and passive exercise are begun. Also, by beginning administration this late we hope to avoid impairment in the healing of tendons.

I am sure you all will recognize the patient who has sweaty, moist, cool palms with perhaps some vasospastic mottling, and who is certain to develop severe complications such as stiffness, neurocirculatory phenomenon and the like following any sort of hand surgery, even, surprisingly enough, following minor surgical intervention. These people develop what I like to call a neurocirculatory asthenia of the hand. We believe that cortisone administration, beginning about three and one-half weeks postoperatively, is extremely valuable in these patients.

Along with cortisone administration, active and passive exercise and all suitable forms of physiotherapy are pushed to the utmost, and it is surprising to what extent even refractory patients can go, or be coerced to go, while cortisone is being given.

JOSEPH L. POSCH (closing): Dr. Weckesser presented an excellent series, with good results in

84 per cent of the patients and subjectively relief of pain in all. Our use of cortisone has been confined mainly to the treatment of an occasional case of Quervain's disease or to injection of ganglion at the distal interphalangeal joint of fingers and infrequently for local instillation in Dupuytren's contracture at the conclusion of the operative procedure.

Experimentally it has been shown that cortisone does not delay healing of tendons, it apparently does depress adhesions and does have an anti-inflammatory activity. Along with the previous clinical and experimental work that was done, Dr. Weckesser has accumulated a series of cases with excellent results.

I think in technic the complimentary incision is extremely important in the palm. Occasionally we have found that with just this one incision in the palm, the adherent tendons in the finger can be freed. We have used fascia rarely around the freed tendon, but found that subcutaneous fat works very well. I think it is an excellent idea to use the polyethylene catheter as Dr. Weckesser does, and to insert the cortisone after the wound is closed. Apparently it is a fairly well accepted fact that the injection of cortisone locally does decrease the pain postoperatively. I noted that Dr. Weckesser mentioned the use of antibiotics postoperatively. We have not been in the habit of using them routinely. His Table 1 is of extreme interest in that two cases obtained improvement (fair in one and excellent in the other), seven years after the original injury. In some of the cases that he has shown we probably would have done a tendon graft rather than a freeing of adhesions. We have performed a tendolysis on twenty flexor tendons following fractured phalanges, with definite improvement in all. In some of these the results were quite remarkable. Of eight cases of tendolysis of extensor tendons, four obtained some improvement, but four were associated with flexor tendon adhesions along with a severe injury to the proximal interphalangeal joint and none of these showed improvement. In five tendon grafts were carried out, with improved motion. The reason we have done tendon grafting procedures on so few is that our policy has been that if a primary repair has been done and limitation of motion results or ensues, a tendon graft is done instead of a tendolysis. I am inclined to think that we probably would have obtained better results with tendolysis if cortisone had been used. It is conceivable that in the future, with the utilization of this drug, the surgeon may sometimes perform tendolysis and obtain good results, rather than do a tendon graft in reconstructive work in surgery of the hand.

Soft Tissue Injuries to the Feet and Legs

MICHAEL L. MASON, M.D. AND JOHN L. BELL, M.D., *Chicago, Illinois*

As in other parts of the body, soft tissue injuries to the feet and legs are divided into two categories, namely, closed and open wounds. Contusions, lacerations, avulsions, crushing wounds and burns and abrasions may occur singly or in combination, and are the subjects of this presentation.

The surgical principles involved in the care of soft tissue injuries to the feet and legs are essentially the same as those of other parts of the body. When disturbed wound healing occurs in leg or foot injuries, however, weeks are often required to secure final closure and disability is greatly prolonged. To insure satisfactory healing even apparently minor injuries often justify a few days of bedrest after adequate surgery. Because of the dependency of the wounds and the frequent concomitant circulatory disturbances, especially in older patients, every effort should be made to obtain primary wound healing.

When confronted with the patient having an obvious injury to the leg or foot, the history of the circumstances of the injury should be recorded, making note of the mechanism of the injury, the time of its occurrence, and what prior care had been rendered. The examiner should inquire as to the patient's general health and complaints. Physical examination to determine the presence of injuries other than the obvious injury to the lower extremity is essential to determine priority of care. Examination may reveal the presence of concurrent injuries or disease processes which might influence the choice of anesthesia or the extent of the initial operative care.

As in hand and forearm injuries, it is essential to evaluate nerve and tendon functions distal to the site of the wound at the time of the initial examination. Any circulatory disturbances should be ascertained prior to operation. Except in superficial lacerations, it is advisable to supplement physical examination with roentgenograms.

Patients with open wounds should receive protection against tetanus infection, using the toxoid booster in patients previously immunized and tetanus antitoxin in patients who have had no previous active immunization. We do not use antibiotics routinely in acute injuries. Adequate surgery is the best prophylaxis against gas gangrene, and the antitoxin for gas gangrene has been overrated.

FIRST AID

The scope of first aid should be directed toward avoiding further injury and contamination, and controlling hemorrhage. Cleansing, removal of foreign material and application of medicaments to the wound as first aid measures are contraindicated except in patients with chemical burns, in which case removal of the offending agent is necessary to avoid continued damage.

Hemorrhage from wounds of the leg and feet can usually be controlled by elevation of the extremity and application of compression over the wound. The routine use of a tourniquet is a dangerous practice. An improperly applied tourniquet may aggravate blood loss by impeding venous return without stopping arterial inflow into the extremity. In such a case every injured vessel is the source of constant outpouring of blood. On the other hand, tourniquets which have been applied with enough pressure to control arterial flow may cause severe damage to the underlying vessels and nerves or may precipitate vasospasm of the collateral circulation of the extremity. If elevation and local compression fail to control profuse hemorrhage, we apply a blood pressure cuff to the thigh with a pressure of 280 to 300 mm. of mercury.

SURGICAL CARE

Contusions. Contusions of the foot and leg accompanied with marked swelling due to hemorrhage should receive prompt surgical care to avoid necrosis. The skin overlying the anterior surface of the tibia is especially vulnerable in that its blood supply may be compromised by an underlying hematoma. In such

instances closed wounds are eventually converted to open wounds. The lateral peroneal muscle compartment is likewise intolerant of swelling, and muscle necrosis has been observed following closed injuries with uncontrolled swelling.

In many cases the application of a resilient compression dressing and elevation of the extremity may be sufficient to control hemorrhage and swelling of the tissues from contusions. We seldom employ aspiration for contusions over the anterior surface of the tibia. If continued swelling or evidence of compromise of skin viability is present, prompt operative intervention is often advisable to avoid necrosis and infection. After surgical evacuation of blood clots and control of hemorrhage, the status of the circulation of the skin should be re-evaluated. Non-viable skin should be excised, and on the anterior surface of the leg it may be necessary to use split-thickness skin grafts or relaxing incisions to allow closure of the wound without tension. Dead spaces must be obliterated to insure survival of skin flaps or skin grafts.

Lacerations. The care of lacerations of the feet and legs does not differ essentially from similarly appearing wounds of the upper extremity. If the wound is contused or avulsed, excision of devitalized tissue is essential to obtain primary wound healing. As in other parts of the body, closure without tension is paramount. The prerequisites for forearm and hand injuries should also prevail for primary repair of divided nerves and tendons of the foot and leg. Unless there is certainty that primary wound healing will ensue, the repair of deep structures should be avoided at the time of the initial procedure.

Loss of function from divided nerves or tendons should be determined by physical examination prior to operation. Peroneal nerve injuries are not uncommon because of the superficial course of the nerve in the upper part of the leg. Division of the Achilles tendon is a disabling injury. Conversely, division of the long extensors of the second, third and fourth toes over the dorsum of the foot is not followed by disability if the short extensor tendons are intact.

Exposure of divided deep structures is aided by a bloodless field, working from normal tissue proximally and distally toward the site of the injury. Accessory incisions are planned to avoid disabling scar contractures. Wherever possible, incisions should parallel the natural skin creases and, as in the forearm and hand, the use of the T incision should be avoided because of the difficulty in obtaining sound closure. For repair of nerves and tendons we employ fine non-absorbable suture material and rely principally upon immobilization and purposeful splinting to keep tension from the suture line of the repaired structures.

Crushing Injuries. Soft tissue crushing injuries over the anterior aspect of the leg are common. The disability from such wounds even in the absence of a fracture can be marked if primary wound healing does not occur. The essential steps in the management of a crushing injury are thorough cleansing of the wound and its surrounding area; excision of devitalized tissue; and, if primary closure of the wound is undertaken, no tension should be employed. It may be necessary to use locally fashioned pedicle flaps or relaxing incisions to make such closure possible. Split-thickness skin grafts should be used to cover the donor area of the pedicle.

Degloving injuries of the leg or foot are usually associated with severe crushing of the soft tissues as well as avulsion, and the inadequate removal of devitalized tissue in order to perform an anatomic restoration usually leads to failure of wound healing and infection.

In the majority of crushing injuries the defects following wound excision are too large to allow closure by wound edge approximation. Unless one is absolutely certain of complete removal of devitalized tissue, the primary application of a cross-leg pedicle flap involves too great a risk. We have used relaxing incisions or adjacent pedicles with more certainty and, in many instances, have used split-thickness skin grafts for temporary coverage. Delayed pedicle flaps to afford padding on weight-bearing surfaces may be necessary if the split-thickness skin grafts do not withstand the efforts of walking.

Burns and Abrasions. The treatment of burns and abrasions of the lower extremities is essentially the same as for burns of the hands and forearms. In the majority of cases we find it difficult to assess accurately the exact depth of the severe scald or flame burn seen within the first few hours after injury. Because of this inherent difficulty all burns are regarded as severe until proved to be otherwise involved.

Electrical burns may be more misleading in that the deep destruction is often more marked than the superficial injury would indicate.

Initially, the burned area is cleansed with soap or a mild detergent for ten minutes and rinsed with sterile water or saline. Removal of loose skin and debris is performed with tissue forceps and scissors. A resilient compression dressing and splint are then applied. As in burns on other parts of the body this primary cleansing and dressing is performed without general anesthesia.

On the fifth or sixth day after injury the dressing is removed to the layer of fine mesh gauze adjacent to the burned surface. If the burn has resulted in a whole-thickness skin loss, surgical excision of the eschar will often permit immediate closure of the excised surface with split-thickness skin grafts. Excision and grafting are facilitated by the use of a bloodless field obtained by applying a blood pressure cuff to the thigh with 280 mm. mercury pressure. In some cases following excision, the surface of the burn may not be entirely prepared for the acceptance of the skin grafts without a few days of moist compression dressings to remove the remaining slough.

Primary excision of the whole-thickness loss followed by immediate application of split-thickness skin grafts has been performed in a few selected cases of well localized burns. However, delayed excision after a few days is more often successful because of the clearer demarcation of non-viable tissue. Deep dermal burns are especially difficult to assess in the early stages.

Difficulty in determining the depth of burned bone is often encountered. Frankly necrotic bone may be surgically excised down to healthy appearing bone and covered with skin grafts. If there is doubt as to the advisability of excision and coverage of burned bone, grafts may be applied to the surrounding areas until bone sequestration has had time to occur. The delayed application of a pedicle flap may be necessary to furnish a protective covering over bone which has been temporarily closed by partial-thickness skin grafts.

If burns are closed as soon as possible, the problem of late reconstructive surgery is often avoided. We have had patients in whom split-thickness skin grafts have served well as permanent coverage on the weight-bearing surfaces of the foot.

POSTOPERATIVE CARE

The harmful effects of edema and motion of injured parts is best avoided by a few days' bedrest for patients with more than trivial soft tissue injuries to the feet and legs. Even in the young adult, the circulation of the lower extremity is much less favorable for wound healing than the upper extremity. In patients with minute trauma we often prescribe the use of crutches for the first forty-eight hours.

Wounds are protected by resilient compression dressings which are applied to avoid edema distal to the wound. In injuries to the ankle and leg the dressing should extend distally to the webs of the toes in order to prevent edema of the dorsum of the foot. Care should also be taken to avoid constriction proximal to the wound. Splints of aluminum or plaster aid in maintaining immobilization and the integrity of the dressings.

In patients at bedrest quadriceps exercises are started within the first few days to lessen the chance of additional disability from such inactivity. An overhead trapeze attached to the bed frame is of assistance in helping the patient move about in bed. After forty-eight hours we usually allow the patient to sit in a wheelchair with the injured extremity elevated on the foot and leg rest.

Unless pain or persistent fever are a cause for concern, the initial dressings are not disturbed for approximately five days. Compression dressings may be necessary for as long as three to four weeks postoperatively in order to assure continued solid wound healing especially when skin grafts and skin flaps have been employed. Ambulation is permitted gradually and the effects upon the progress of the wounds are noted.

Ligamentous Injuries about the Ankle Joint

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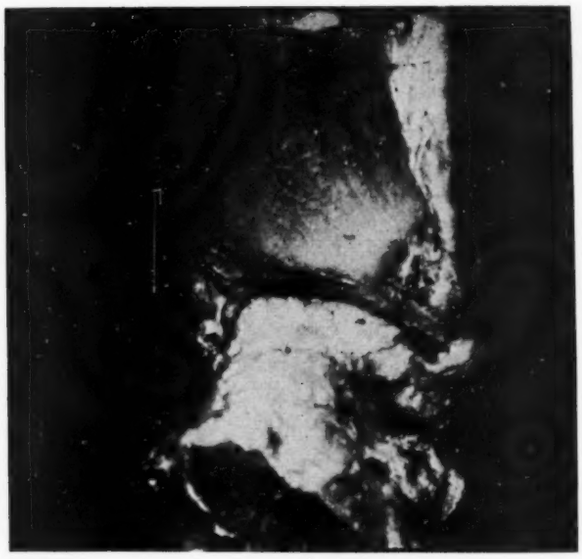
IT is well established that the severity of an ankle injury is directly proportional to the degree of instability present in the ankle following injury. Stability of the ankle is dependent upon two factors: (1) anatomic configuration and (2) ligamentous support.

The distal end of the tibia together with the external malleolus forms a mortise which accurately accepts the convexity of the talus and allows for hinge motion in this joint. (Fig. 1A.) The increased width of the anterior portion of the talus can be received into the mortise only at the expense of a small degree of external rotation of the fibula which occurs at the inferior tibiofibular joint. This anatomic mortise is maintained by ligamentous support.

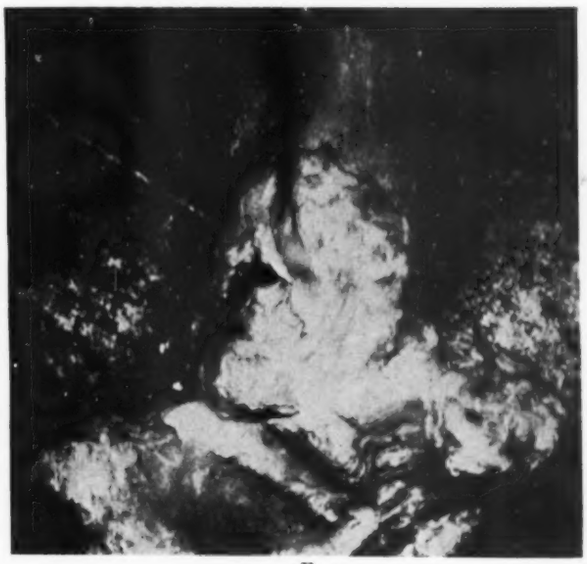
The ligamentous structures about the ankle joint fall into three groups: (1) the medial collateral or deltoid ligament, (2) the lateral collateral ligament and (3) the inferior tibiofibular ligament. The deltoid ligament (Fig. 1B) is a thick, strong, triangular-shaped ligament which originates from the entire border of the medial malleolus and extends anteriorly to insert into the side of the talus, distally to insert into the sustentaculum tali, and posteriorly to insert into the medial tubercle of the posterior process of the talus. Actually, these three components of the deltoid ligament are fused into one, so that the deltoid ligament represents a single ligament. In contrast, the lateral collateral ligament is composed of three separate and distinct fasciculi. (Fig. 1C.) The anterior talofibular fasciculus is a weak band of fibers intimately associated with the anterior capsule of the ankle and extending from the anterior border of the malleolus to the neck of the talus. The calcaneofibular fasciculus is a moderately strong band of fibers which extends from the tip of the malleolus distally to a tubercle on the os calcis which is just proximal and posterior to the peroneal eminence. It differs from the anterior fasciculus in that it is clearly separated from and outside of the joint capsule.[4] The posterior talofibular fasciculus (Fig. 1D) is probably the strongest component of the lateral ligament and passes from the digital fossa of the fibula to the lateral tubercle of the posterior process of the talus. This latter fasciculus also is intimately associated with the capsule and is not readily visualized at operation since it is within the joint. Perhaps the least understood of all of the ligaments of the ankle region is the inferior tibiofibular ligament. (Fig. 1D.) It maintains the integrity of the inferior tibiofibular syndesmosis which is so essential for a normal ankle mortise. The inferior tibiofibular ligament has an anterior and a posterior component, each of which consists of fibers which pass downward and outward from both the anterior and posterior surfaces of the tibia to the corresponding tubercles on the anterior and posterior surfaces of the external malleolus. These two components are reenforced by the interosseous ligament which consists of multiple, short, oblique fibers uniting the apposed triangular surfaces of tibia and fibula.

Watson-Jones[2] has stated that "The former classification of joint injuries into sprains, dislocations, and fracture-dislocations has no good foundation in theory or in practice." This statement is based on the fact that no differentiation is made between simple sprain and avulsion of a ligament. In the one instance a simple sprain involves only the stretching of capsular fibers which permits no instability of the joint, whereas in ligamentous avulsion there is invariably some degree of instability of the joint. When fractures are present, the severity of the injury is apparent and good treatment is instituted. Ligamentous injuries, on the other hand, are less apt to be recognized and as a result inadequate treatment is rendered.

A knowledge of the mechanism of injury, careful clinical examination and correlation with x-ray films will frequently lead to a diagnosis of severe ligamentous injury. Nevertheless, there are those cases in which the x-ray



1A



1B

FIG. 1A. The normal ankle mortise maintained by the medial, lateral and inferior tibiofibular ligaments.

Fig. 1B. The three components of the deltoid ligament fused into a single, strong ligament.

1C

1D

FIG. 1C. The lateral aspect of the ankle showing two of the three separate fasciculi—the anterior talofibular and the calcaneofibular ligaments. The posterior talofibular ligament cannot be seen.

FIG. 1D. Posterior aspect of the ankle showing the strong talofibular fasciculus. Also, the posterior tibiofibular ligament.

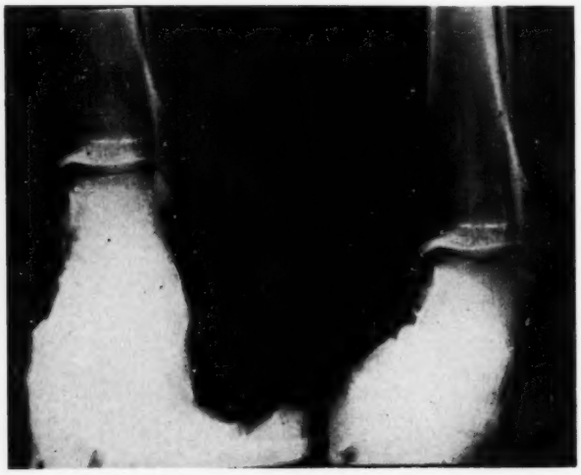


FIG. 2. Stress roentgenography in both eversion and inversion with a tilt of the talus resulting from the inversion force.

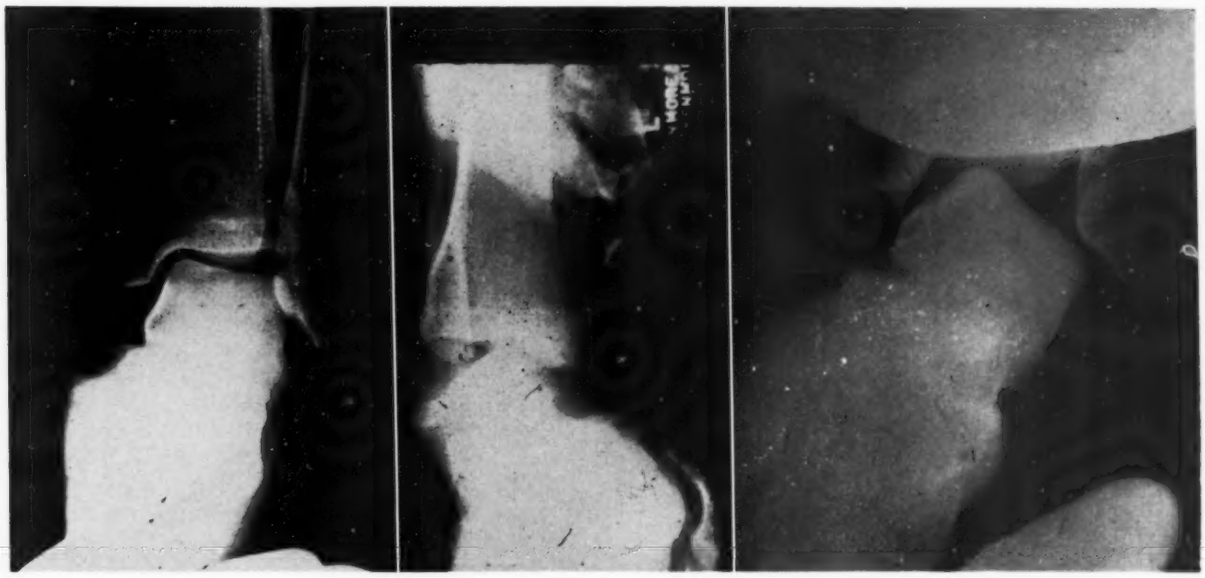
FIG. 3. Comparative views with inversion stress showing normal hypermobility on the left and marked tilt of the talus, the result of ligamentous injury on the right.

findings are misleading and it is then necessary to resort to the use of stress roentgenography (Fig. 2) in order to exclude the possibility of ligamentous injury.[2] These stress studies are adequately performed under local anesthesia using a 1 per cent procaine solution infiltrated into the tender areas about the lateral and medial aspects of the ankle joint. In the occasional case general anesthesia may be necessary to allow sufficient force to be applied to the injured ankle. Stress is applied in both eversion and inversion, with the foot in moderate plantar flexion and x-rays taken in the anteroposterior view while the stress is being applied.

According to Bonnin,[3] a variable degree of mobility exists in the normal ankle and it may allow a tilt of the talus of as much as 5 to 15 degrees. This amount of tilt constitutes the hypermobile ankle, but fortunately the condition is usually bilateral and so can be differentiated from hypermobility resulting from injury which is confined to the injured ankle. Comparative views of the ankle are indicated in questionable cases of hypermobility. (Fig. 3.)

The treatment of injuries to the ankle depends upon the presence or absence of instability, and the amount of instability depends upon the ligament or ligaments ruptured.[3] The anterior talofibular ligament is the most commonly injured ligament following an inversion strain.[5] Rupture of this ligament permits tilt of the talus through 5 to 15 degrees.[3] (Fig. 4A.) When this lesion is associated with rupture of the calcaneofibular ligament, the tilt of the talus is increased to 15–30 degrees.[3] (Fig. 4B.)[3] If, as infrequently happens,[4] the injury involves rupture of all three ligaments on the lateral aspect of the ankle (Fig. 4C), the rotation is as high as 45 degrees; and in this position the lateral convexity of the talus may become locked against the inner aspect of the lateral malleolus.[3]

Our experience indicates that a tilt of no more than 15 degrees can be properly treated by immobilization of the leg in a circular plaster extending to below knee level with immobilization maintained for a period of five weeks. Stress x-rays after removal of the plaster usually show a stable ankle. (Fig. 5.) When the tilt exceeds 15 degrees, it is our impression that the lateral aspect of the ankle should be exposed and, where possible, the torn ligaments accurately repaired. Suture of the anterior talofibular ligament and capsule offers little difficulty; but when the calcaneofibular ligament is also avulsed, it is not infrequently found to be displaced superficial to the peroneal tendons. (Fig. 6A.) It is necessary, then, to retract these tendons anteriorly (Fig. 6B) before a proper insertion of the calcaneofibular ligament can be made. It is difficult to repair the posterior talofibular ligament because of its location posteriorly within the joint capsule, but fortunately it is only infrequently torn. Repair of the anterior talofibular and calcaneofibular ligaments has resulted in normally stable ankles. Following suture of the ligaments, the leg is immobilized in a circular plaster which



4A 4B 4C

FIG. 4. A, rupture of the anterior talofibular ligament with 15 degree tilt of the talus. B, combined rupture of anterior talofibular and calcaneofibular ligaments with 30 degree tilt of talus. C, rupture of all three components of the lateral collateral ligament with 45 degree tilt of talus.

extends to the mid-thigh level with the knee in 20 degree flexion. In this way torsional strains are eliminated from the ankle region during the healing period.

Avulsion of the medial collateral ligament (Fig. 7*) is the equivalent of fracture of the medial malleolus; and because one cannot be sure of the relationship of the torn ends of the ligament, one can draw a further analogy to fracture of the malleolus with interposed soft tissue. It is our belief that all avulsions of the medial collateral ligament should be repaired surgically. In some instances it is possible to resuture the ligament to the remains on the edge of the malleolus; in others, however, it is necessary to make drill holes through the end of the bone in order to effect a good approximation of the strong ligament. Accurate suture of the ligament restores the mortise so that further treatment directed to the lateral aspect of the joint is unnecessary.

Rupture of the inferior tibiofibular ligament resulting in significant diastasis at the inferior tibiofibular joint occurs infrequently as an isolated lesion, yet is seen quite commonly in association with other injuries about the ankle joint. (Fig. 8A.*) The correction of the associated injuries, either suture of the avulsed deltoid ligament or screw fixation of the medial malleolus, frequently approximates fibula to tibia at the inferior joint. However, where widening of the ankle mortise persists, it is necessary to explore the inferior tibiofibular joint in order to remove interposed soft tissue

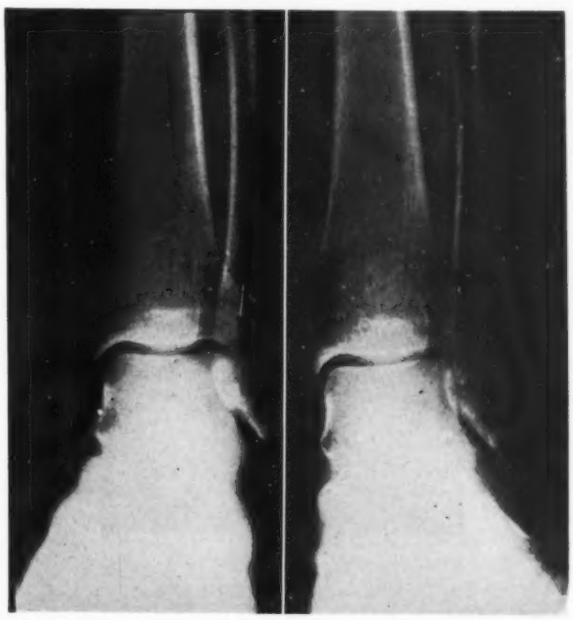
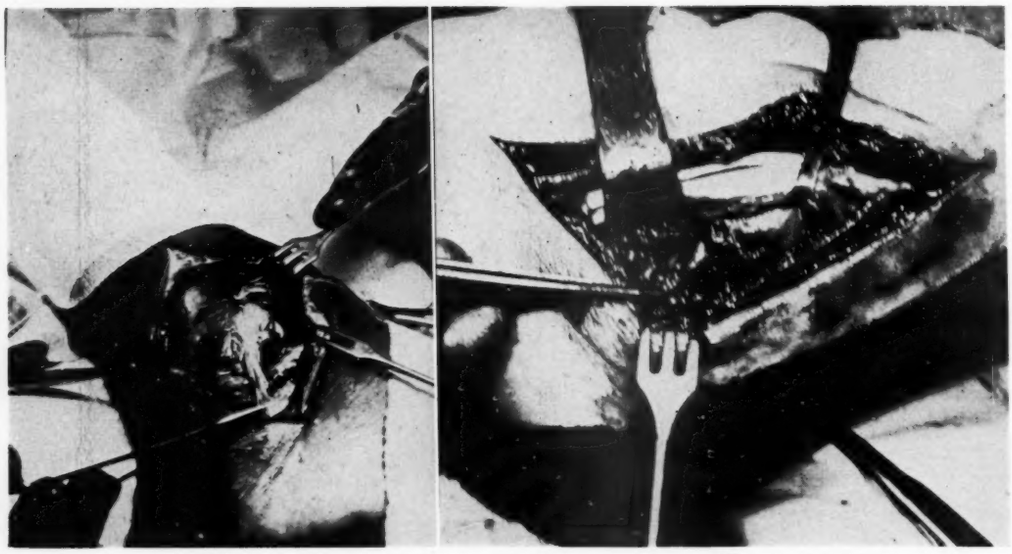


FIG. 5. Inversion and eversion stress x-rays following five weeks' immobilization in below-knee circular plaster. See Figure 4A for inversion stress film immediately following ankle injury.

* Case of Dr. E. Hanavan, Buffalo, New York.



6A 6B

FIG. 6. A, combined tear of anterior talofibular and calcaneofibular ligaments with the calcaneofibular ligament displaced superficial to the peroneal tendons. B, anterior retraction of the peroneal tendons to allow replacement and suture of the calcaneofibular ligament.

FIG. 7. Complete avulsion of deltoid ligament associated with fracture of lateral malleolus.

8A 8B

FIG. 8. A, rupture of inferior tibiofibular ligament with avulsion of fragment of anterior tibial tubercle and associated with fractures of medial malleolus and lower shaft of fibula. B, restoration of mortise by screw fixation of medial malleolus and bolt through tibia and fibula to stabilize the inferior tibiofibular syndesmosis.

and sometimes to fix this joint with a bolt through fibula and tibia. (Fig. 8B.)

SUMMARY

The severity of an ankle injury is directly proportional to the instability of the ankle mortise. Stability is dependent upon: (1) anatomic configuration and (2) ligamentous support.

The deltoid ligament is a single ligament as contrasted with the three separate fasciculi of the lateral ligament.

Stress roentgenography is necessary to exclude the possibility of ligamentous injury.

Tilt of the talus of less than 15 degrees is treated by plaster immobilization of the foot

and leg; tilt of the talus greater than 15 degrees is an indication for suture of the torn ligaments.

All avulsions of the deltoid ligament should be repaired surgically.

Surgical treatment directed to the inferior tibiofibular joint is indicated only when the mortise remains widened.

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DISCUSSION OF PAPERS BY DRS. MASON AND BELL, AND DZIOB

John J. Cranley (Cincinnati, Ohio): My work is such that I am apt to see patients five to twenty years following injury to the lower extremity when ulceration of the leg has developed. In reconstructing the story, a very typical history is obtained. From the time of injury there has been daily swelling of the ankles. This swelling subsides during the night but recurs during the day. After a period of time the skin of the lower leg becomes pigmented and hard, and finally ulceration occurs.

This is the typical "postphlebitic" ulcer. Phlebitis has almost certainly occurred at the time of injury but has been unrecognized. It is easy to appreciate why it is unrecognized, because the patient is expected to have swelling and pain from the injury. If there has been a fracture, the patient is expected to have some residual swelling which is usually ascribed to a loss of tone of the soft tissues.

There is another type of patient who has a minor injury to the leg with a break in the skin. The patient may have had no evidence of venous disease before or after the injury, yet this small ulceration of the leg does not heal or heals much more slowly than an ulcer of similar size would on another portion of the body.

In my opinion, both these types are due to one fundamental factor, that is, an increase in ambulatory venous pressure or a relative increase in gravitational pressure in the limb due to the patient's inability to walk normally and thus pump the blood and fluid out of the leg by muscular activity. Either type of ulcer can be healed in all instances by elevation and in almost all instances by the patient's wearing a pressure bandage while ambulatory. However, the difficulty is that how much elevation or how much pressure is needed is not widely appreciated. When we speak of elevation, we mean elevation of the limb above heart level; and when we speak of a pressure dressing on the leg, we mean sufficient pressure to overcome the hydrostatic pressure of the blood. The ordinary cotton elastic bandage simply does not provide enough pressure. A rubberized one must be used, and in some instances a second bandage must be applied over the first in order to get sufficient pressure to overcome the hydrostatic pressure and eliminate all swelling.

The practical aspect for prevention of these ulcers is to insist that the patient wear a heavy enough rubber bandage or elastic stocking to eliminate all swelling during the period of convalescence from the injury to the leg. It is believed that this will prevent formation of the postphlebitic ulcer at a later date.

Harrison Lloyd McLaughlin (New York, N. Y.): Dr. Dziob is to be complimented for calling attention to an eventually crippling lesion, the unrecognized rupture of the lateral ligaments of the ankle. This lesion is easily and frequently overlooked when it is an isolated injury, but rupture of either medial or lateral ligaments is also prone to pass unrecognized if the examiner allows his attention to be distracted by the presence of an obvious contralateral malleolar fracture. The injured ankle with roentgen displacement of a single malleolar fracture also contains a ruptured ligament subjacent to the other intact malleolus.

The advisability of anesthesia and stress films in every case of sprained ankle seems open to question. It has been our practice to follow this method only in the presence of swelling and ecchymosis sufficient to indicate gross subcutaneous tissue damage. Documentation of the diagnosis has been an indication for operation. In late cases direct repair has proved impossible and the ligaments have been replaced by tendon grafts. In early cases direct repair has proved difficult and insecure, and has usually been supplemented by the implantation of tendon grafts.

Joseph M. Dziob (closing): I would like first of all to thank Dr. McLaughlin for his kind words and to admit that it was because of a presentation which he made some three to five years ago, in which he brought out the fact that ligamentous injuries about the ankle could be severe, that I was stimulated to carry on stress roentgenography to uncover what had been an enigma to me up until that time.

He disagreed with the statement that, "Where gross fractures exist in association with ligamentous injury, adequate treatment is usually given." I am assuming that the individual who recognizes the gross fractures in the ankle joint treats those fractures according to accepted principles. In other

words, if one reduces a fracture of the medial malleolus so that in the lateral view there is no suggestion of interposed soft tissue and then applies a circular plaster, I am quite sure that the end result will be a good, serviceable ankle. There may have been an associated ligamentous injury on the lateral aspect of the ankle which was unrecognized, but the treatment rendered for the medial malleolus fracture often suffices to allow healing of the torn ligaments. I am still going to stick to the principle that where there is bony injury the end result is much better than where ligamentous injury exists alone, only because the ligamentous injury is less apt to be recognized.

Do we take stress films of every sprained ankle? In the last three years of conference in the Buffalo area I have been accused of gross exaggeration many times because I answer that stress studies are performed in our Clinic for practically every sprained ankle. However, I think that these same accusers are beginning to accept our plea that they carry out a similar mode of diagnostic treatment. We believe that it is almost impossible to differentiate simple sprain from ligamentous injury without the use of stress studies.

Dr. McLaughlin states that he has trouble suturing the torn ligament on the lateral aspect of the ankle joint. I am a bit surprised about that because I have seen him perform surgery on shoulders which were really much more difficult, and he handles those very well. If the ligamentous injury around the ankle joint is approached early, there is no difficulty whatever in suturing the torn anterior talofibular ligament with its associated capsular tear. However, the calcaneofibular ligament is sometimes difficult to locate because of its normal position deep to the peroneal tendons. In some instances the calcaneofibular ligament need only be replaced in the normal channel and it will rest against the tubercle on the lateral aspect of the os calcis.

What happens if the torn lateral ligaments are not sutured? Time alone prevented us from showing slides which depict clearly the result of inadequately treated torn lateral ligaments. Clinically, the ankle was unstable and stress studies showed an abnormal tilt of the talus. At operation there is little remaining ligamentous tissue and it is necessary to perform some type of tenodesis operation in order to replace the torn, atrophied ligaments.

Use of Deep Procaine Injection for the Relief of Localized Pain*

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ADAMS[1] recently stated that "pain, in its severer forms, as for example, trigeminal neuralgia, migraine, or the various colics, is one of the heaviest crosses the human race has to bear—persistent pain, whether mild or severe, over weeks, months or years can produce serious and far-reaching sequelae which affect the patient's work, play and sleep, alter their personalities and produce changes in every phase of their daily lives." He goes on to emphasize that "until recent years physicians as a whole tended to minimize the importance of persistent pain, particularly, if the cause was not readily apparent." In discussing Adams' paper Dr. R. H. Barrett says, "The sympathetic nervous system is a dominant factor in pain. Except for direct nerve trauma, ischemia or localized hypoxia, it is probably the basis of most pain."

The character of pain in the segmental neuralgias[2] caused by irritation of the extraspinal or paraspinal elements that constitute the segmental distribution may vary from stabbing, tearing, shooting or cramp-like to a milder aching, sore, drawing, pulling, dragging type of pain. Painful areas which are localized "and are tender to the slightest touch, or brushing of wearing apparel or bed clothes, and are associated with burning suggest a causalgic type of pain which is sympathetic in origin." The term atypical neuralgia is used to differentiate it from segmental neuralgia on the one hand and to indicate its sympathetic component on the other.

As a history of trauma cannot be elicited in all cases of headache, migraine, occipital neuralgia, neuralgia of the chest simulating angina pectoris, backache and sciatica, postherpetic neuralgia and pain from sprains along the costal margin and xyphoid, the term atypical neuralgia would be more appropriate than Livingston's[3] "post-traumatic pain syndrome" such as occurs in whiplash injuries[4] or in musculofascial pain.[5]

Livingston[3] mentions atypical facial (or buccal)[6] neuralgia to call "attention to the fact that trigger points may develop in the face, neck and scalp just as they do in the extremities or in the back; that they seem to be capable of initiating pathologic reflexes as do other trigger points, and the fact that they sometimes yield to simple measures suggests that irritation from peripheral sources may play some part in the development of pain syndrome involving the head area."

Peripheral nerves[7–9] have been shown to contain, besides somatic fibers, sympathetic motor (vasomotor, sudomotor, pilomotor) and afferent sympathetic fibers conveying pain impulses.

If one considers the location of pain in headaches, the trigger points, which intensify the pain on compression, are frequently found as a point of deep tenderness beneath the skin of the scalp, or in the occipital and neck muscles and their fascia, or at the ends of the transverse processes of the cervical vertebrae or beneath the superficial layer of skin. (Figs. 1 and 2.)

In describing the method of injecting trigger spots with 2 per cent procaine, I wish to emphasize the most accurate identification and painstaking injection of the sensitized areas. The trigger points are located by making pressure first with the rounded end of a ball pen and then its tip, progressing tangentially toward maximum sensitivity. The trigger points are rechecked several times, making pressure in the same direction, and finally are marked so that when the fine No. 25 needle with a very short bevel is introduced, exactly the same direction is followed as with the pen. The deposition of 2 per cent procaine is begun just beneath the skin and made gradually deeper until all trigger

* This paper was Read by Title at the meeting of The American Association for the Surgery of Trauma.

spots have been reached. Usually from 2 to 5 cc. are injected in each area and not more than 40 or 50 cc. are injected at a visit.

Hematologic studies have been found to be normal in these patients who bruise easily and since a hematoma at the site of injection

FIG. 1. The cross marks indicate the location of the usual trigger points. (Modified from Sobotta's Atlas of Human Anatomy, Philadelphia. W. B. Saunders Co.)

vitiates the success of the treatment, 10 mg. of vitamin K are injected subcutaneously before treatment and ½ gr. of phenobarbital is given by mouth at least twenty minutes before the injection is started.

No patient receives an injection of procaine, no matter how small in amount, without first receiving a barbituate which reduces the toxicity of procaine about five times. Patients who are known to be sensitive to procaine receive ½ gr. of phenobarbital orally three hours, two hours, one hour and one-half hour before treatment. An ampul containing 2 gr. of sodium phenobarbital is always available, half to be given intramuscularly if a patient should have a reaction to the 2 per cent procaine. Adrenal cortex extract, ½ cc. subcutaneously, is also given.

Many of the patients have learned that alcohol will, in the course of two to six hours, cause a return of their headache or pain. It is felt important to instruct all patients about the probable effect of taking alcohol in any form after injections.

The long-continued pain has furnished the alarm reaction, as described by Selye,[10] with depletion of the patient's adrenal cortex in particular, and frequently also the anterior pituitary, as evidenced by their excessive perspiration or tendency to cry easily.

In restoring impaired endocrine glands such as the anterior pituitary and adrenal cortex, a

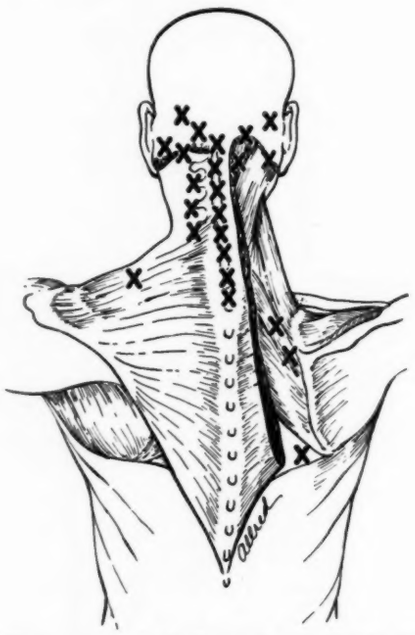

FIG. 2. The cross marks indicate the usual trigger points. The vertical cross marks are between the cervical spinous processes where the torn ligamentum nuchae makes a "bowstring." (Modified from Sobotta's Atlas of Human Anatomy. Philadelphia. W. B. Saunders Co.)

high protein, high vitamin C diet is essential. Multivitamins and minerals are given. Since salicylates interfere with the absorption of vitamins (and many patients were taking salicylates for pain) the digestive enzymes, pancreatin and bile salts are given before a meal with the vitamins, along with a 3 gr. suprarenal cortex tablet. For exhaustion, one or more suprarenal cortex tablets are given during the day, and for perspiration or weeping tendency, one or more 5 gr. anterior pituitary tablets are sucked until relief is obtained. Potassium chloride, 15 gr., is given daily to protect the cellular potassium from the effects of these hormones. Calcium is given in some form by mouth or injection to reduce the excitability of the neuromuscular apparatus. Since June, 1954, I have found that the tendency to cry or to have profuse perspiration could be relieved by the action of the Rauwolfia root or its alkaloid, reserpine.[11] This substance at least

partially suppresses the sympathetic predominance at the hypothalamic level.

Migrainous headaches[12] may come in attacks initiated by fatigue, menstruation, food idiosyncrasy and other exciting causes. The underlying neurologic mechanisms which produce hemicrania and other disturbances such as hemianopsia, paresthesia, lacrimation and nasal congestion indicate involvement of the sympathetic nervous system.

In occipital neuralgia a history of soreness or stiffness in the neck with limitation of motion may precede or be associated with the headaches which usually begin in the occipital area, radiating to the frontal or supraorbital region. The headaches are steady and vary from a mild ache to a severe throbbing bursting pain worse on stooping or straining at stool. At times the frontal headache is so predominant that painful symptoms referable to the occipital region or neck are submerged, but sensitive areas are readily formed across the occiput or along the spines or transverse processes of the upper cervical vertebrae. (Figs. 1 and 2.)

When the pain from sensitized spots of the head has been sufficiently long in intensity and duration, a painful contraction of the skeletal muscles of the head, neck, shoulders and even the erector spinae group in the lumbar region may develop and produce further involvement.

After trauma other than sprains from a whiplash injury, such as slipping and trying to prevent a fall, the occipital and neck muscles may be strained; and after certain infections, notably virus diseases, influenza and rheumatic fever, the sympathetic nervous system can become sensitized. An injury may be produced such as a strain to the cervical paravertebral ligaments, to the fascial attachments of the short rotator capitis muscles, to the vertebral processes or to the trapezius and rhomboid attachments to the vertebrae and scapula. Tenderness between the cervical spines or near their transverse processes is frequently found. (Fig. 2.) Another source of irritant sympathetic nerve fibers is in the interspinous ligament localized between the vertebral spinous processes. (Fig. 3.) Trauma to the head and neck, often having occurred in childhood and long since forgotten, may tear loose the ligamentum nuchae from the cervical spinous processes. When the head is bent forward this shredded ligament forms a bowstring from the occiput to the last cervical or first thoracic spine. Trigger points are to be found in this ligament or deeper between the spinous processes or at the ends of the ligament. (Fig. 2.)

Studies by Rinzler and Travell[13] in 1948 on the somatic component of cardiac pain have shown that in some patients with angina

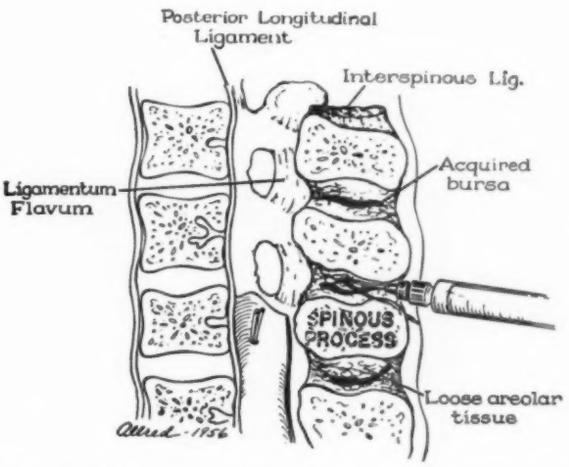

FIG. 3. The location of the ligamentum flavum, the interspinous ligament with its bursa. The needle for procaine injection penetrates between the spinous processes into the interspinous ligament and bursa, down to the transverse arch or the ligamentum flavum. (Modified from J. C. B. Grant's Atlas of Anatomy. Baltimore, 1947. Williams & Wilkins Co.)

pectoris and acute myocardial infarction pain is relieved by blocking secondary trigger areas in the skeletal muscles of the chest. They emphasize[14] that the main source of irritation in the pain syndromes involving the chest are sprains and strains at the origins of the pectoralis major and minor muscles, and sometimes at their insertions as well. (Fig. 4.) That these thoracic pain syndromes[15] do occur makes their recognition important, otherwise invalidism may result or a diagnosis of psychoneurosis may affect the patient's entire outlook on life. Trauma of strains, long forgotten, may have been the irritant initiating the pain cycle, and illness with concomitant vitamin and endocrine imbalance may so increase the patient's susceptibility to pain that the syndrome develops. Thirty patients with such pains have been referred for surgical relief of cervical herniated disk or for thoracic sympathetic interruption for angina. A few internists have recognized the syndrome as a neuralgia and have located trigger spots which intensify the pain upon compression.

In a report on the treatment of post-thoracotomy neuralgia in ten patients,[16] the most important trigger points for injection with 2 per cent procaine are into the interspinous ligaments at least two above the intervertebral processes and two below where the rib cage was

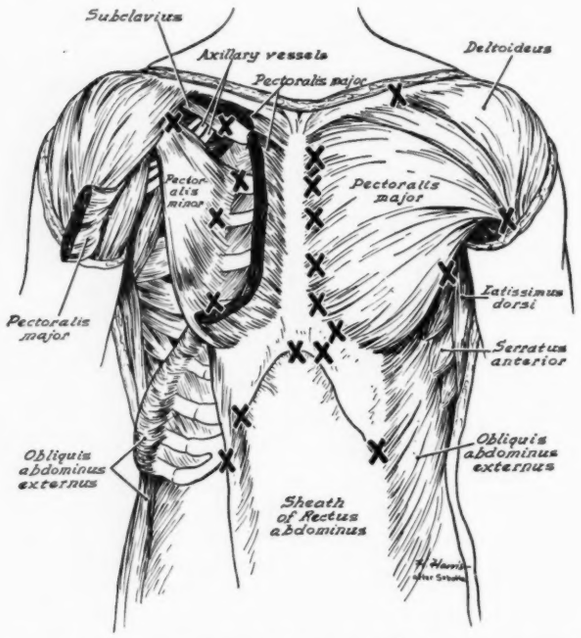

FIG. 4. The cross marks indicate the location of the usual trigger points in the atypical neuralgias of the chest simulating angina pectoris. (Modified from Sobotta's Atlas of Human Anatomy. Philadelphia. W. B. Saunders Co.; Reichert, F. L., *Am. J. Surg.*, 88: 399, 1954.)

forcibly separated. Strained or traumatized origins and insertions of the pectoralis major and minor muscles are additional important trigger areas, as is also the xyphoid.

Some thirty patients comprising the costal margin and abdominal group are of interest because, again, their complaints, often occurring in paroxysms of pain lasting from two to four days at a time, had been puzzling from a diagnostic viewpoint. Again, the continuance of the pain over the years prompted them to seek relief with the result that many negative gastrointestinal series of roentgenograms, as well as intravenous and retrograde pyelograms, had been made. As this pain was usually in the upper abdomen, either epigastric or in the right or left upper quadrant, referred at times to the right and left back, it was diagnosed as acute biliary colic, renal colic, chronic duodenal ulcer, splenic infarction, recurrent pancreatitis or acute intestinal obstruction. Tenderness was present and at times rigidity could be elicited. However, in the absence of fever and leukocytosis, exploration was rarely done.

On examining these patients during their attack, the pain and tenderness is actually at

FIG. 5. Incisions carried down to deep structures to divide sympathetic fibers involved in postherpetic neuralgia. (Reichert, F. L., *Stanford M. Bull.*, 13: 357, 1955.)

the xyphoid[17] or along the costal margin and not deep in the abdomen. The tip and either side of the xyphoid, as well as the costoxyphoid junction, may have numerous trigger points which when extremely accurately located and pressed upon elicit the identical pain of which the patient complains. (Fig. 4.) The irritating lesion is probably from scar compression following the sprain or strain of the attachments of the rectus abdominis, internal oblique or transversus abdominis fascia to the costal cartilages and xyphoid. Slipping during bowling or other activities, jarring or falls during horseback riding or other accidents, or compression of the tissues by retractors in exposure during an operation may have been the traumatic irritant.

Low back pain, with or without sciatica, has been attributed to dental caries, a misplaced uterus, osteoarthritis, slipped sacroiliac articulation and herniated intervertebral disk. Favorable reports in the literature are based on the correction of these etiologic factors. Certainly, muscle spasm seems to play a part no matter what may be considered the etiologic factor. Very frequently immediate relief has been

produced by injecting procaine into trigger points in the muscles or muscular attachments to the transverse processes of the vertebrae, the ilium and sacrum, to the trigger points along the sacroiliac junction and into the lumbar and upper sacral interspinous ligaments.

The location and surgical removal of a herniated intervertebral disk in the lumbar region is assured when, neurologically, findings are obtained of segmented numbness of the leg and foot with absent or diminished reflexes and definite motor weakness in the foot. If the neurologic examination shows numbness with normal reflexes, or diminished or absent reflex without numbness, I have found it wise to first inject procaine into the interspinous ligaments from L1 to S2 and also to inject the sensitive spots of the sacroiliac articulation. In fourteen patients immediate relief of pain occurred with these injections. The numbness was gone or the reflex returned to almost normal. If the pain returned in a day or two, and again on repeated reinjection (five patients), a herniated disk was suspected and removed surgically.

Herpes zoster, or shingles, causes burning pain even before vesiculation. After the vesicles have dried, the pain either subsides or becomes chronic, persisting for months or years. This postherpetic neuralgia,[18] supposedly from a viral infection involving the sympathetic ganglia, is a constant burning pain exaggerated by light touch or movement of the affected part. These patients are excellent examples of the effect of stress on the body, and in the past three years much attention has been paid to restoring depleted endocrine glands, especially the adrenal cortex and anterior pituitary, as well as in treatment of this neuralgia. In our experience when the neuralgia involved the trunk or chest, nine patients have had complete relief, two had slight relief and one was not helped by blocking the appropriate sympathetic ganglia with a 2 per cent procaine or a similar anesthetic oil (bromsalizol®). Frequently, even after ganglionic blockage, local trigger points are found which respond to procaine injections. In postherpetic "trigeminal" neuralgia, blockage of the fifth nerve or its branches gives no relief but injection of the stellate, first and second thoracic sympathetic ganglia in some instances gives relief. In the other neuralgias a more peripheral approach to the sympathetic fibers, either by injection or by division of tissues and vessels,[19] will afford some relief if not cure. (Fig. 5.) Of nine patients with neuralgia involving the "trigeminal" distribution, six have been completely relieved and three are greatly improved.

SUMMARY

The injection of 2 per cent procaine into accurately located trigger points has in general brought relief to patients suffering from chronic pain or atypical neuralgia.

Chronic pain or atypical neuralgia is due to irritation of peripheral sympathetic fibers found in subcutaneous tissue, muscles, fascia of muscles, about blood vessels and in peripheral nerves, attachments of muscles to bony structures and in the para- and interspinous vertebral ligaments.

The atypical neuralgia occurring in headaches, occipital neuralgia, neuralgia of the chest simulating angina pectoris, post-thoracotomy neuralgia, backache and sciatica, postherpetic neuralgia and sprains along the costal margin and xyphoid furnishes the alarm reaction with stresses that frequently deplete the patient's adrenal cortex and anterior pituitary glands in particular.

Continuance of relief after procaine injections of the trigger points seems to depend on abstinence from alcohol and restoring to normal the vitamin, mineral and endocrine imbalance.

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The Medical Clinician and Disability Evaluation

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THE first compensation acts were established in this country about forty-five years ago and two principal objectives were initiated to benefit the injured laborer: (1) freedom from the uncertainties of common law litigation, thereby relieving a burden on public charity and (2) subsistence payments to tide the victim over the period of wage losses during recovery from injury.

The latter objective has been accomplished very well, but litigation still demands major judicial responsibility in the settlement of claims for total and partial permanent disability. No improvement seems to have been found for the provisions in the compensation laws which designate specific awards based on amputation values. Since the residuals of injury often result in partial permanent disability other than that of amputation, litigation is necessary to evaluate the disability.

The services of the medical profession are of essential importance in the administration of justice under the compensation laws. The responsibility of the medical profession is essentially twofold. (1) The treatment and rehabilitation of the injured worker must be determined and (2) the extent of injury to the body and the residual disability limiting the worker in pursuing his livelihood must be determined and evaluated.

In this panel discussion we are concerned with this second phase of the medical responsibility which involves the doctor's opinion on the extent of permanent partial disability. Unfortunately this field of medical practice is lacking in scientific fundamentals. In the first place, the schedules for disability found in the statutes are not based on scientific deductions. The present compensation laws are but a prototype of the early enactments which established specific awards for amputations at various levels, which is a compromise for the sake of expedience. From the medical point of view the scientific significance of physical disability cannot be measured by monetary values established on the basis of amputation. What then is the medical examiner to do other than to compromise on scientific fundamentals in evaluating disability under the rules of the court?

If the statutory award for amputation of an arm at the shoulder is specified at 50 per cent of permanent total disability, can the doctor's opinion that this is a greater or lesser disability be of any value? How should back or head injuries be measured? Can a back injury be 75 per cent of total permanent disability when it would not be considered nearly as disabling as the loss of an arm at the shoulder, which has a statutory value of only 50 per cent? Can a fracture deformity of the forearm be rated as a 50 per cent loss of use of the hand when it is not as severely disabling as the statutory value of amputation of the thumb which is considered only 30 per cent loss of the hand? Should two injuries of a similar nature result in any greater disability compensation in the state of Oklahoma than in the state of New York? Judging by the aforementioned, is it not time that the medical profession establish scientific fundamentals upon which disability could be evaluated?

The subject is too extensive in scope and far too complex for the average clinician to comprehend. A vast medical knowledge of such subjects as anatomy, physiology and clinical diagnosis is necessarily precluded. It often involves, as well, fundamentals of biomechanics, biochemistry, physics, geometry and mathematics. Where the true static, kinetic and dynamic alterations of the structures of the human body are concerned, how can the clini-

cian be an expert scientific witness if he has no greater ability for medical analysis than that of looking at an x-ray of a bone or joint deformity, observing the history and clinical findings and rendering only an arbitrary opinion as to the extent of disability? Is he not likely to be subject to false impressions and influences beyond customary medical reasoning?

Inasmuch as the disability awards provided by legal enactments are more or less arbitrary, it has been suggested that a comprehensive schedule of ratings for all grades of disabilities be established by a medical committee. The American Medical Association is considering such action. This, no doubt, would be of utmost value to the busy clinician who has no time to go into the finer details of the medical analysis of disability.

The necessity still remains for a practical method of making a comprehensive medical analysis of all the elementary factors of disability upon which to arrive at a dependable percentage of permanent disability. Since the legal enactments for total and partial disability are arbitrary values and since the medical evaluation of disability is for the benefit of the court in establishing a loss of earning capacity, the medical opinion can be little more than a thorough analysis of all the factors bearing on the ability of the disabled person to continue working for his livelihood. Why not, then, apply the clinical observations to a system of medical analysis which will give a thorough over-all average percentage of loss as compared to normal? By such a method each and every factor must be evaluated for the percentage less than normal. What is the percentage of functional loss to the body or parts of the body? The anatomy has been altered to what percentage? The clinical symptoms indicate body deficiency to what percentage? To what extent of normal percentage are the readjustment and reactions to job restoration handicapped? Is it not true that a careful analysis of all these factors and an average of the percentage values would give a satisfactory answer to the disability problem?

The author has advocated a system of this kind for many years.* About the only objections that have been raised are those relating to the difficulty in understanding the method and the amount of time it requires. The fundamental principles of medical responsibility cannot be compromised. The answer is either right, wrong, or not applicable. A brief statement of this method of analyzing disability follows.

Disability is divided into two principal components, namely, (1) loss of function and (2) loss due to physical disorders in relation to work capacity.

Loss of Function. The loss of body function must be analyzed and evaluated independently on movement and action of the injured parts of the body. The disabled body, or parts of the body, should be determined for loss of function by the following: (1) quickness of action, (2) security and confidence, (3) coordination and skill, (4) strength, (5) endurance, (6) safety and (7) influence on re-employment.

Physical Disorders in Relation to Work Capacity. The extent of actual mass damage to the anatomy is determined by clinical examination, x-ray and other laboratory procedures. An evaluation of the actual anatomic damage as compared to the normal is decided upon.

The clinical manifestations are thoroughly investigated and weighed with the history and subjective claims of the patient. A percentage is then established as an evaluation for the abnormal versus the normal.

The restrictions of disability in relation to physical requirements at work are analyzed. The extent to which activity is to be restricted in lifting, pulling, standing, crawling and kneeling should be determined as should all other activities which must be applied to normal work. The percentage of loss is then decided upon.

The restrictions of working conditions and a lowered tolerance for environmental influences are considered. Can the patient work in outdoor exposure? Is he likely to be affected by dampness? Will he be endangered by certain machinery hazards, slippery floors, and the like which are found in average working conditions? The percentage of depreciation is decided upon.

The reactionary interferences influencing return to work, or the intangible factors altering future endeavor to work, are applied to the disability evaluation. The age of the person, the adverse reaction of the employer to re-hire, seniority rights, limited opportunities for re-

* McBride, E. D. Disability Evaluation, 5th ed. Philadelphia, 1953. J. B. Lippincott; American Academy of Orthopaedic Surgeons Instructional Course Lectures, vol. 12, January 1955; *J. Internat. Coll. Surgeons*, 24: 1955.

habilitation, the likelihood of repeated exacerbations of disabling pain, and many other similar intangible factors should be weighed as to their influence on the disabled person as compared with his normal state. A percentage of loss is then established.

The final percentage of disability may be obtained by taking an average of all the percentages of loss decided upon under the various divisions of analysis. An average of the percentages of each of the physical disability factors and of the functional factors will give an average overall percentage of disability. It is the author's opinion that the functional factors of loss are actually more important than the applied physical disorders, so that the average percentage of the functional factors may be weighted by .75 and the average percentage of the physical disorders factors weighted by the remaining .25. The formula then can be stated:

Per cent Average Function Loss × .75
+ Per cent Average Physical Disorders
× .25 = Per cent Disability

It is safe to say that if an existing disability is analyzed in the manner described there will be a conscientious feeling of thorough investigation and the percentage of evaluation will not be far from right.

A chart has been devised for the convenience of keeping a case record of the disability evaluation. Any reader interested in this chart may contact the author.

Disability Evaluation

ALFRED H. WHITTAKER, M.D., *Detroit, Michigan*

To investigate the problem of disability evaluation the President of the American Medical Association appointed a committee in 1953 composed of Alexander Aitken, Earl McBride, Alfred Whittaker and Henry Kessler. Henry Kessler acted as chairman and Carl Peterson as secretary.

The instructions to the committee were to study the subject, and to determine if a need for more comprehensive study was indicated. Then they were asked to outline the significant information gathered, to state the conclusions arrived at, to establish methods of evaluating the factors entering into the problem and to put this information into a publication to be distributed to physicians in general practice and in the specialties in which evaluation is frequently involved.

The committee met regularly, did reach certain conclusions and recommended that a primer or guide on disability evaluation be printed and made available to all physicians. Following an organizational meeting the committee first studied the history of disability evaluation and the philosophy and methods of evaluation in our own society as well as that in other governmental states. Workmen's compensation was studied first since it effects medicine primarily and since the most intensive evaluation and rating has been carried out in this area.

At the 1953 Convention of the International Association of Industrial Accident Boards and Commissions the medical committee presented a report which reflected current confusion. The committee pointed out that the last serious attempts to discuss this controversial subject took place in 1923 and 1928. So much criticism and hard feeling and so many different ideologies arose that further discussion was abandoned. In reviewing the literature of these early controversial years it was evident that the main issues and basic concepts were clouded by senseless comparisons between states and provinces. In the ensuing thirty years there has been an industrial and economic revolution resulting in marked advances in labor relations but, despite this progress in allied fields, little, if any, change has occurred in workmen's compensation laws and administration of these laws, particularly in permanent disability evaluation.

The medical committee of the Industrial Accident Boards and Commissions went on to state that critical analysis of some of our laws shows there is still a tendency to buy off the injured workman, placing emphasis on monetary values for certain injuries. Until and unless there is a change in our basic thinking, evaluation for permanent disability will still remain the jumbled, chaotic mess it is.

Our committee found that this is an era of Social Security in which four states already have compulsory disability laws and in which all states have workmen's compensation laws, so that every unemployed person presents a potential problem to the medical profession. A large portion of the population of this country are covered by voluntary plans of hospital and medical care and this care is provided on a doctors certificate of temporary or permanent disability. Pre-employment and periodic examinations, now a requirement of medical departments in industry, and placement studies, pension plans, advancement by seniority, forced retirement, early request for retirement and requests for employment past the retirement age all shift a great responsibility to the physician. The United Nations at its tenth anniversary, in appraising its accomplishments, pointed out the emphasis being placed on human rights. These rights are an integral part of the health and physical condition of the people of fifty-eight nations and only the doctor can interpret disability. Medical officers in the overseas armed forces must pass upon the physical condition of applicants for marriage. Under a new ruling of the commissioner of baseball no ball player can be placed on the inactive list without a doctors certificate. In the courtroom doctors have been playing an important part in judicial proceedings, the

determination of the extent of damages depending in many instances directly upon the medical testimony which is in itself a disability evaluation.

Medical certification under state compulsory disability programs is an example of the increasing importance of disability evaluation. It is doubtful that physicians other than those resident in the four states where the laws are in operation have any sort of working knowledge of this type of coverage or its implications to the medical profession; yet the medical certification of disability is the heart of the program.

Today we are discussing medical problems of workmen's compensation laws, yet all of our states have such programs. In the beginning, starting from the time Wisconsin adopted its law in 1911, the medical profession was unfortunately totally indifferent to the workmen's programs. Not until medical provisions were introduced did the medical profession enter the picture.

Now, when we come to compensation for non-occupational disability, the problem becomes even more complicated. The physician is faced with a personal conflict in which emotion necessarily takes precedence over reason. To the physician not experienced in disability evaluation, the broad definition of disability lends itself to many interpretations regarding diagnosis, sickness and injury and evaluation thereof. With the recognition of responsibility within the profession comes cooperation between the administering agencies and the medical profession, a step best demonstrated when the California law was instituted.

Unfortunately disability evaluation is not an exact science resting on exact rules inevitably resulting in the same conclusion irrespective of the person operating the formula. As Chester C. Schneider has pointed out, the conclusions reached vary with the evidence and concomitant significance, the previous experience of the physician and the condition and reaction of the patient. Appraisal of injuries and the disability potential is a complicated and important subject. It requires extensive knowledge of all diagnostic procedures in order to evaluate the nature of the injury and the probable consequences in terms of healing and degenerative changes.

Trauma to the human organism which may require disability evaluation may be divided into four categories: (1) no objective evidence of injury and no underlying defects or disease; (2) no evidence of injury but underlying defects or disease of long standing; (3) evidence of injury and no associated antecedent defects or disease, and (4) evidence of injury and associated long standing defects or disease.

It must be conceded that pain and disability can exist without visible physical anomalies. Injuries which disturb the circulation of important structures often lead to slowly evolving defects which cannot be detected for months or years after the accident.

Disability evaluation does not involve simply the mechanical measurement of restriction of motion and function, but should take into consideration also the concurrent psychological, economic and social problems involved. Even disabilities of the hand present far reaching effects upon the human organism, and upon the ability of the worker to earn a living and to make social and psychological adjustments. A responsibility also exists to the private and governmental agencies involved.

Factors involved in disability evaluation are: (1) trauma to the structures; (2) effects upon the human personality; (3) economic status of the injured; (4) Social Security status of the injured; (5) recognition of the problem by the attending physician; (6) attitude of rehabilitation agencies; (7) attitude of governmental and social agencies, and (8) attitude of management and of governmental agencies toward employment of the handicapped.

Forces resulting in disability are: (1) Trauma: Terminal disability is never static and can be overemphasized. It involves deformity, malunion, loss of tissue, disfigurement, change of contour, psychologic changes, loss of dexterity, interference with locomotion, incoordination, loss of one or more senses, loss of perspective, fatiguability, postural deformity, loss of joint motion and contractures. (2) Disease: Gangrene and amputation or contracture; intracranial disease with loss of function of extremities, spinal cord degeneration and infection with musculoskeletal impairment. The end result of rheumatic diseases is rheumatoid arthritis, osteoarthritis, fibrositis, gouty arthritis, infective arthritis. Neuropathic disease would also come in this category. (3) Constitutional Biologic Inadequacy: A hereditary status resulting in the inability to withstand the wear and tear of modern living and

is characterized by anatomic change, susceptibility to disease and emotional imbalance and psycotic states with varying degrees of disability. (4) Radiation Injuries: An increasing number of disability states have been caused by released energy such as the x-ray or nuclear fission which result from the mass release of radioactive waves and particles. Effects may vary from superficial burns to severe blast injury and include dermatitis, neoplasia, damage to blood-forming organs, cataracts, impaired fertility and genetic defects. (5) Psychologic Imbalance Due to Trauma: It is recognized that physical trauma may result in defects of the psyche with varying degrees of disability. Although numerous wars have left countless maimed and injured, "traumatic neurosis" did not appear as a clinical entity until the latter part of the nineteenth century. The almost sudden appearance of this ailment in Europe and America can be correlated with the existence of insurance. Reichardt in his exhaustive study says over and over again that neurosis may follow trauma, but is not caused by it. His thesis reflects the view that neurosis is psycogenic, a desire to receive compensation. Since we are working with large numbers of people influenced by the insurance factor, this reaction must be considered in all cases of disability evaluation.

Palmer has remarked that a speedy evaluation of injury insured elimination of uncertainty on the part of the patient, a more rapid recovery and an earlier return to work. It is axiomatic that the earlier the injury is competently handled surgically, the better is the prognosis for ultimate function. Less axiomatic but equally true is the statement that the earlier both temporary and possibly permanent disability are evaluated, the better the chance of orienting the patient for future capacity.

No problem in disability evaluation can be solved by a study of the localized area of injury; the problem should be approached from the standpoint of the "whole person." The final decision reflecting local disability and its effects upon the human organism must be based on the relationship of the injured person to the world in which he must continue to exist.

Studies have shown that a large percentage of industrial, traffic and home injuries are cared for by the general practitioner. Many industrial injuries occur in small plants which have no full time industrial surgeon and which are near offices and general hospitals.

Thus there is an urgent need to acquaint the medical profession with the fundamental principles involved and the proper procedures to be carried out in establishing accurate and unbiased disability evaluation.

Disability Evaluation in Workmen's Compensation

ALEXANDER P. AITKEN, M.D., *Brookline, Massachusetts*

IN this discussion I shall confine my remarks to disability evaluation as currently practiced in workmen's compensation cases. In industrial medicine physicians have been searching for many years for some means of evaluating disability. Some answer is apparently required, not as an aid in treatment but to meet the demands of laymen that are involved in the legalistic aspects of injury. Commissioners of industrial accident boards, insurance carriers and attorneys for plaintiff and defendant insist, for the purposes of adjudication, that the physician resolve the patient's disability into mathematical terms. It is obvious that identical injuries may produce severe disability in some persons while producing none in others and may result in serious handicaps in performing some occupations and be of little significance in others. The physician, aware of the numerous tangible and intangible factors involved in disability and desirous of being fair to all parties, often finds resolution on this basis difficult if not impossible. Even with the improbable adoption of a uniform compensation act for each of the forty-eight states, it seems highly unlikely that any formula could ever be developed which could be applied equitably to all persons and still include such factors as age, physical condition, work potential, occupation and work availability, to name a few.

Why do we find ourselves in such a dilemma? Forty years ago many types of injury did indeed result in permanent disability. Medical science had not developed to the point where it could offer much in the way of physical restoration. Social conditions were such that the disabled worker and his family became a liability to society. Even when able to do some work the disabled man had great difficulty in securing employment. The compensation acts existing at that time provided for some indemnification for the worker for his anatomic loss but the awards were too niggardly to be of practical value.

Today, with the progress of medicine and the development of antibiotics, we no longer see the disabilities of forty years ago. With the development of physical rehabilitation and vocational training, it is questionable whether any injury is truly permanently and totally disabling, provided the injured person is given every opportunity and has the fortitude to rehabilitate himself. We can be proud of our accomplishments in reducing disability, although we still have much to learn regarding adequate medical care after injury and the use of physical rehabilitation and vocational training.

Although the compensation acts have become far more liberal they have not kept pace with sociologic changes or with the progress of medicine. Weekly compensation rates in some states still fail to provide for adequate family security. The disabled worker still has as much difficulty securing employment as he did forty years ago. The provisions in many states fail to allow for adequate and continuous medical care and no provision is made for physical rehabilitation or vocational training. We are still being confronted with indemnity based on local anatomic or functional loss and the presence of disfiguring scars. Little or no attention is paid to the injured workers genuine need, namely, that of restoration to gainful employment. Questions of disability are still settled by laymen and the merits of a case are defended by laymen in the courts or through legal procedures of our industrial accident commissions. In these legal entanglements the physician is but a pawn. He must not consider the physical, psychologic, domestic and financial complications of the injury; he must resolve the disability into percentages much as one would appraise the damage of an automobile. In the end an adjudication is reached. The commissioner, frequently untrained for his job and confused by the welter of conflicting medical testimony, clears the case from his docket. The insurance carrier is relieved of further liability. The attorney receives his commission and the doctor his fee. The patient gets his award, a sum of money which he spends

and a disability which he keeps. Such a system invites legal controversy and invites the prolongation of disability for financial gain long after the disability has ceased. However, it is no wonder that a truly disabled worker, realizing that his work potential and market value have been jeopardized, will prolong the disability until he has received his award. In this he is often aided and abetted by his legal counsel whose interest in the award is not altogether altruistic. How is it possible for any physician with a clear conscience to render a disability evaluation in those states in which the law specifically limits the maximum medical expenditure to $200 or limits the duration of medical care to ninety days? Disability evaluation under these circumstances is a fraud. The injured worker must be given an opportunity, through adequate and continuous medical care, to restore himself to gainful employment. He must not be deprived of these rights through unrealistic and antiquated compensation laws.

It is high time the medical profession took stock of the entire situation. It is time we evaluated our responsibilities to the injured worker and fulfilled our obligations toward restoration. And we must assume a dominant role in demanding that the responsibilities of others, namely, labor, management, insurance company and government, be fulfilled. A cooperative effort has already made considerable progress under the aegis of the Subcommittee of Industrial Relations of the Committee of Trauma of the American College of Surgeons. This committee, in cooperation with the C.I.O., A.F. of L., U.M.W., the United States Department of Labor, the United States Public Health Service, Office of Vocational Rehabilitation and two insurance companies, has been studying for the past five years the problems of the injured worker. An article on the basic principles for rehabilitation of the injured worker and another on the operating principles for a modern workmen's compensation system have been developed and approved by the Regents of the College. I should like to quote these as I feel they are pertinent to today's discussion.

PRINCIPLES FOR REHABILITATION OF INJURED WORKER

1. Rehabilitation of the injured worker and his return to gainful employment should be the basic concept in an improved workmen's compensation system. We recognize that the disabled worker wants to be rehabilitated and restored to gainful employment. Settlement of cases on a basis of cash awards alone does not meet the continuing needs of the injured worker and his family.

2. The need for higher standards and broadened benefits in workmen's compensation is recognized. Such standards and benefits should be developed against the background of presently known advances in physical restoration and vocational rehabilitation and adequate standards of individual and family need.

3. Full utilization of all our potential manpower is essential to the welfare and strength of the country at all times. The discarding of disabled workers is an economic extravagance detrimental to the welfare of our country, wholly aside from the personal effect on the worker and his family. Solution of the problems of trauma requires co-operation, and not competition, between all interested groups and agencies. We must therefore improve and expand all activities, public and private, that aid in rehabilitation of the disabled worker.

4. The medical profession should adopt the concept that the responsibilities of the treating physician extend over the entire period of disability to the end that the patient is restored to gainful employment at his highest attainable skill.

5. Rehabilitation and restoration to gainful employment of the injured worker must begin with first aid and continue through the period of disability. In order for a physician to carry out this responsibility, it is essential for him to recognize the total medical problem of the patient in addition to his injury, as well as his personal problems. The physician must bring to bear on these problems all the skills and disciplines that science and society can offer, and utilize all community resources which can assist him in the accomplishment of these objectives.

OPERATING PRINCIPLES FOR A MODERN WORKMEN'S COMPENSATION SYSTEM REQUISITE FOR THE ATTAINMENT OF THE PRINCIPLES FOR REHABILITATION OF THE INJURED WORKER AS ESTABLISHED BY THE AMERICAN COLLEGE OF SURGEONS

I. Before the principles for rehabilitation of the injured worker as established by the Subcommittee on Industrial Relations of the American College of Surgeons can be attained a critical review of the current practices in workmen's compensation, medical care, and medical and vocational rehabilitation is required.

II. Since rehabilitation of the injured worker and his return to gainful employment is one of the basic concepts of workmen's compensation, changes in the attitude, laws and administration of this system are essential.

A. It must become the duty of the compensation agencies to supervise the medical care of workmen's compensation cases with the view:
   1. To determine the accuracy of the medical diagnosis.
   2. To see that competent and continuous medical care is provided as long as medically indicated.
   3. To see that adequate medical and vocational rehabilitation is provided when indicated.
   4. To make sure that an injured worker has obtained the maximum recovery possible before making any final financial determination of permanent disability.

B. To assist the compensation agencies in the performance of these duties, panels of impartial medically qualified experts should be established within the compensation systems. These panels should be utilized in the performance of the following functions of the agencies:
   1. Supervision of all medical care to insure the adequacy and continuity of medical care from the date of injury or disability, whether due to accident or occupational disease, to maximal restoration.
      a. To review and examine when necessary all seriously injured cases at an early date to determine:
         (1) The need of consultant services by medical specialists.
         (2) The need of medical rehabilitation.
         (3) The disabled person's work potential and the need for vocational rehabilitation.
   2. To establish standards for the provision of medical rehabilitation services to workmen's compensation cases.
   3. To determine the causal relationship of injury or disease in contested cases and the need of additional panels of impartial medically qualified experts for such determination if necessary.

C. Attainment of the basic principles can be accomplished only by changes in the administrative rules of procedure and/or in the compensation laws themselves. Provisions must be made for:
   1. Complete and continuous medical care from the date of injury or disability, whether due to accident or occupational disease, to maximal restoration without statutory limitation of cost or duration.
   2. Complete medical rehabilitation, referral to vocational rehabilitation services, and adequate financial support for such services until maximal restoration is achieved. Appropriate and medically prescribed return to work before maximal restoration is an accepted rehabilitation procedure and may also be carried out as on the job training or retraining.
   3. Adequate compensation to insure family security during the entire period of disability and rehabilitation.
   4. Expansion of compensation statutes to broaden second injury fund provisions to further encourage industry to employ physically handicapped workers.
   5. Coverage of all employees including those of small establishments and those engaged in occupations now considered as non-hazardous.

   To insure the maximum efficiency in administration the law should require the appointment only of experienced individuals as commissioners. These positions should be career appointments at adequate salaries and not subject to changes in administration.

   III. The costs of the vocational rehabilitation of the disabled worker should be borne either by industry or the State vocational rehabilitation program or jointly.

   A compensation system so constituted would restore more efficiently and completely far more individuals to gainful employment and would at the same time eliminate the present highly controversial and expensive procedures and in the end would be far less expensive to industry, labor, the insurance carriers and the community as a whole.

Under such a system, the present litigious miasma would be eliminated. Vast numbers of injured workmen would be restored to useful employment at their highest attainable skill. Many workers and their families would be free of the stigma of dependency on public welfare. A need for evaluation exists, of course, but disability would be determined by medical men, in the light of the way in which it affects the person and his overall ability to return to useful employment.

If this Society has pent-up energy which seeks an avenue of productivity, I hope it will follow the lead of the American College of Surgeons in striving for more realistic compensation laws. We should not entrench the present archaic system by attempting to develop methods of disability evaluation which allow others to slough off their responsibilities or to make financial gain at the expense of the injured workmen.

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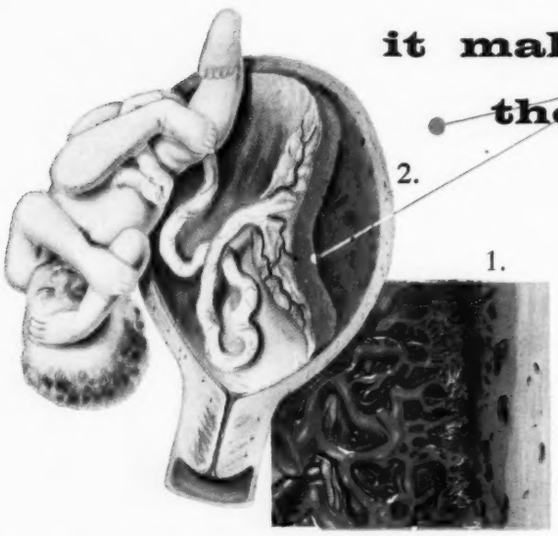

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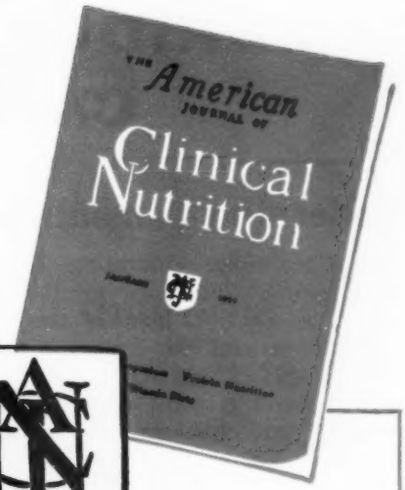

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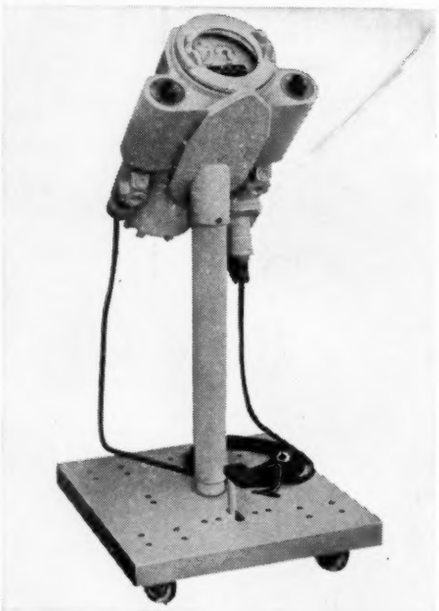

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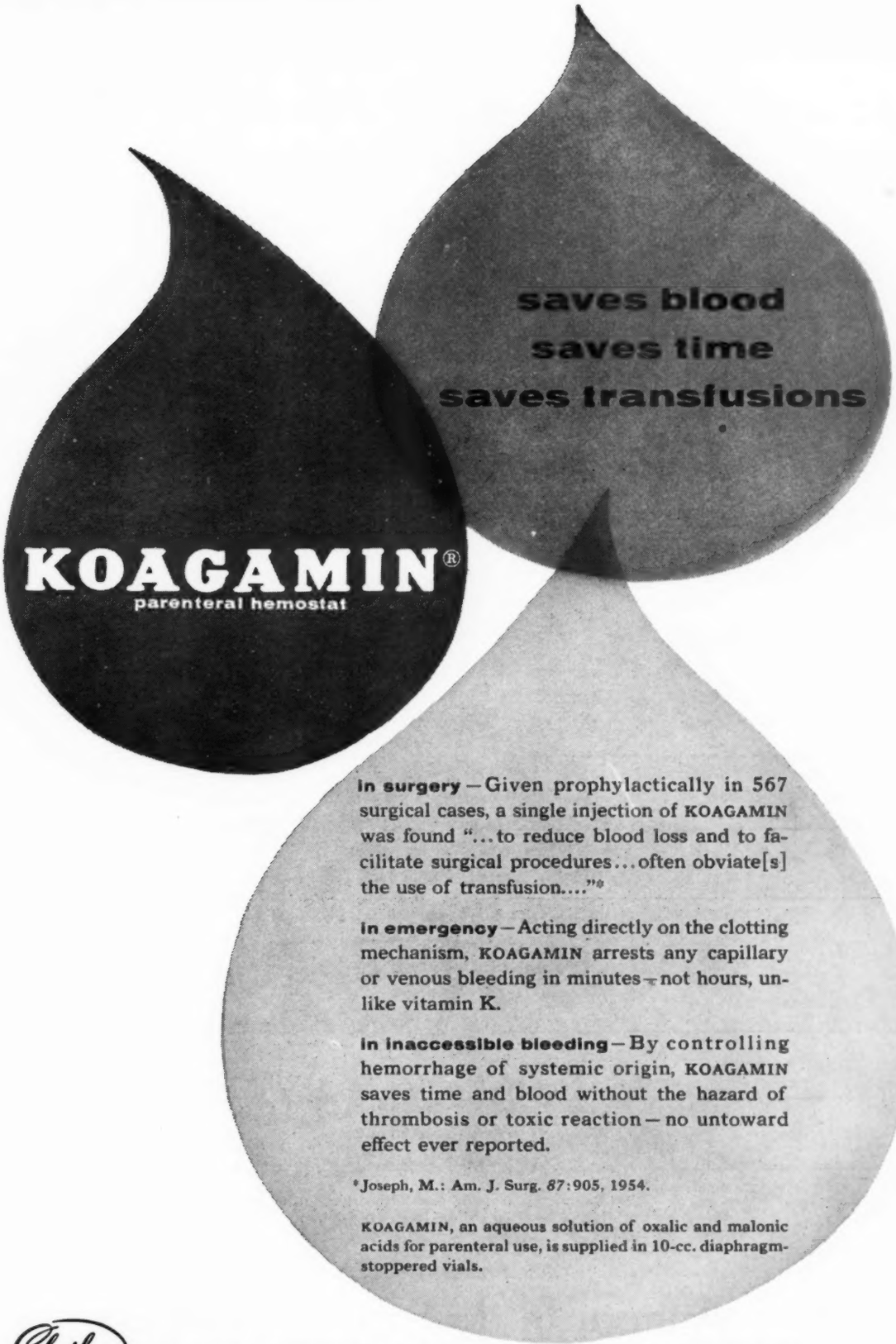

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*Joseph, M.: Am. J. Surg. *87*:905, 1954.

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Meat...

and Sodium-Restricted Diets

The therapeutic usefulness of sodium restriction in congestive heart failure, cirrhosis of the liver, hypertensive disease, certain affections of the kidneys, and the toxemias of pregnancy makes it essential to have authentic data available on the natural sodium content of foods, both fresh and processed.* This information enables physician, dietitian, and patient to choose foods proper in kind and amount to assure a sodium intake within the prescribed limits. Another essential consideration is the nutritional adequacy of the sodium-restricted diet.

For the nutritional protection of the patient, especially when treatment must be prolonged, the sodium-restricted diet must not compromise the provision of adequate calories, protein, and other essential nutrients. In order to assure adequate top quality protein, sodium-restricted diets frequently employ considerable amounts of animal-derived protein foods, such as meat, since meat is notably high in protein but only moderate in sodium content. For diets extremely restricted in sodium, canned meats are available from which most of the sodium has been removed.

Severe limitation or omission of meat jeopardizes an adequate intake of protein, iron, thiamine, and niacin. Therefore, sodium-restricted diets which fulfill allowances for all nutrients recommended by the National Research Council, yet furnish only 500 mg. of sodium, may include 5 ounces (155.5 gm.) of meat (fresh basis, equal to about 4 ounces when cooked).* For easy reference, the table below sets forth the sodium content of various kinds of meat.

Sodium Content of Meat (fresh)*
(mg. per 100 Gm.)

Beef, lean, muscle	70	Lamb, lean	90
Beef heart	85	Pork, lean	55
Beef kidney	200	Pork liver	80
Beef liver	130	Veal	100
Beef tongue, unsmoked	80		

*Davidson, C. S.; Clifcorn, L. E.; Clifford, P. A.; Gabuzda, G. J., and Robinson, C.: Sodium-Restricted Diets, the Rationale, Complications, and Practical Aspects of Their Use, Division of Biology and Agriculture, National Research Council, Washington, D. C., Publication 325, 1954.

The nutritional statements in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

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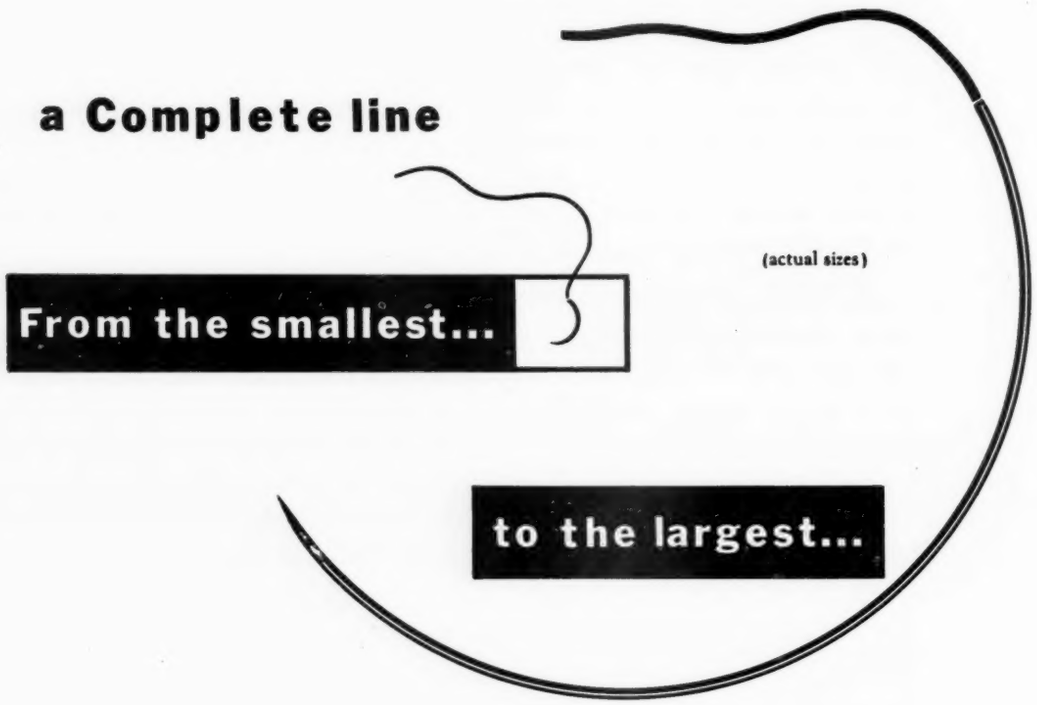

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References: 1. N.N.R. 1954, p. 107. 2. J.A.M.A. 153:1516 (Dec. 26) 1953.

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1. Grayzel, H. G., Heimer, C. B., and Grayzel, R. W.: New York St. J. M. 53:2233, 1953.
2. Heimer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951.
3. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Ind. Med. & Surg. 18:512, 1949.
4. Turell, R.: New York St. J. M. 50:2282, 1950.

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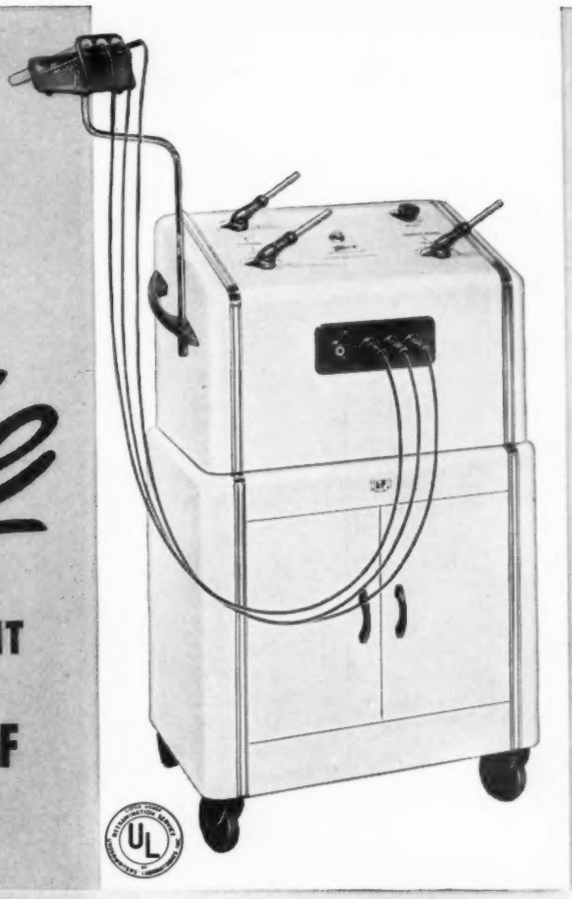

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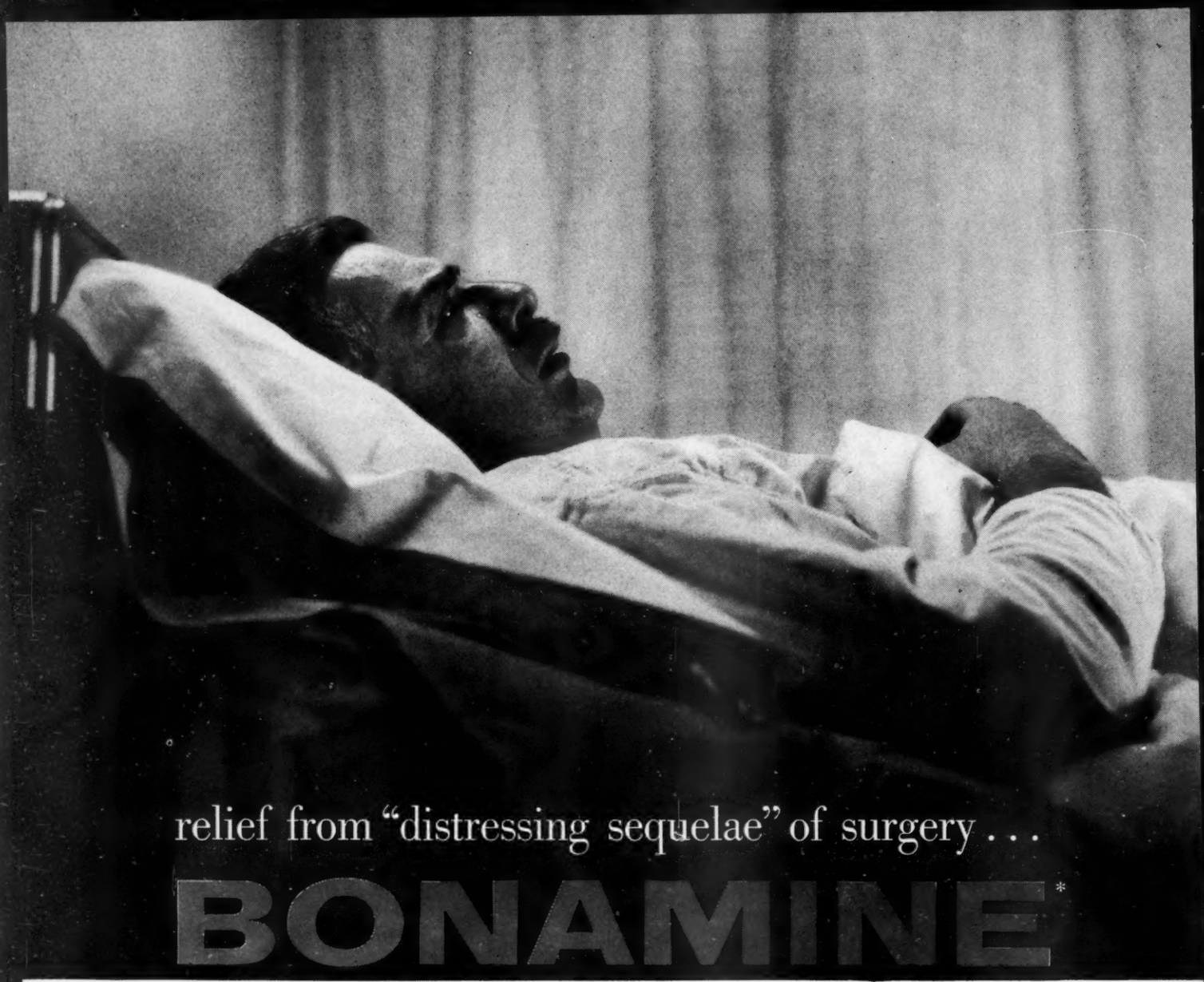

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Rubin, A., and Metz-Rubin, H.: Surg., Gynec. & Obst. 92:415 (April) 1951.

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1. Scribner, B. H., et al., Fluids for Parenteral Administration, New Eng. J. Med., 252:443-445, 1955.

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1. Gross, J. M., Jl. Int. Coll. Surg., 23:24, '55.

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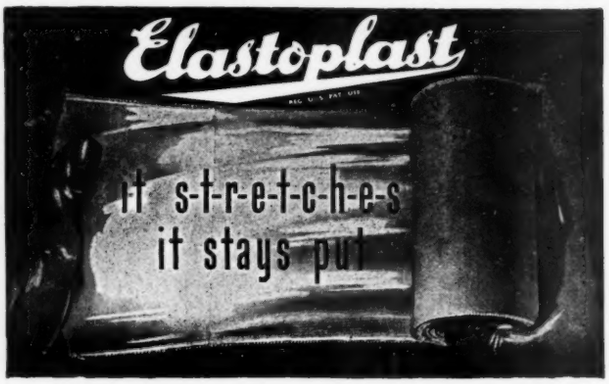

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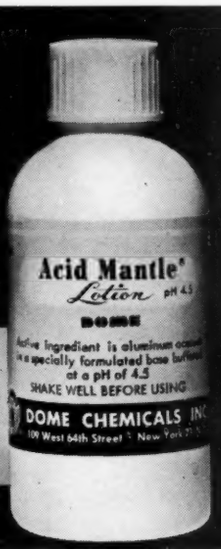

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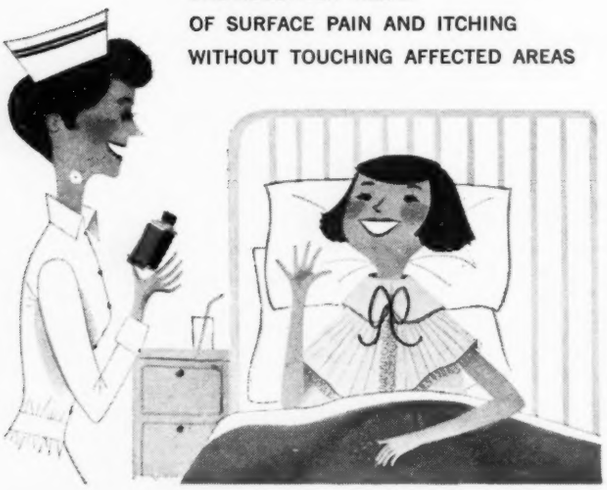

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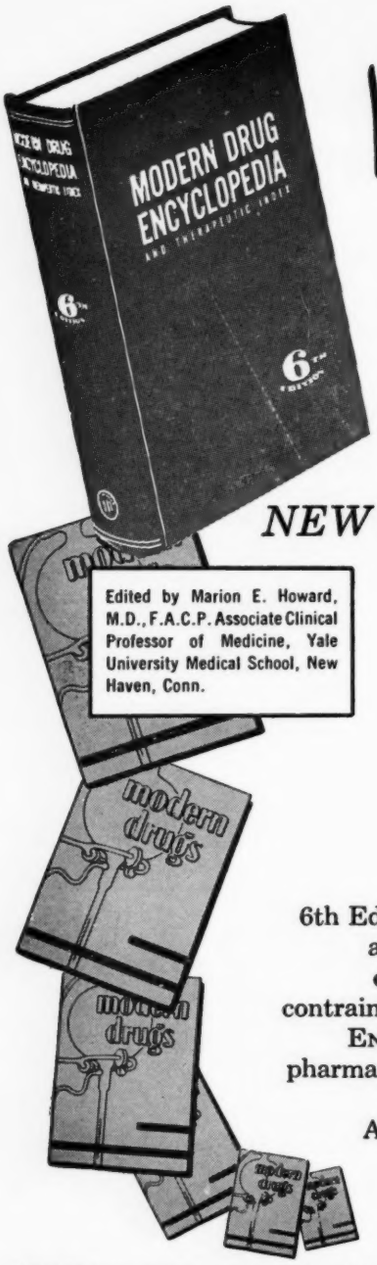

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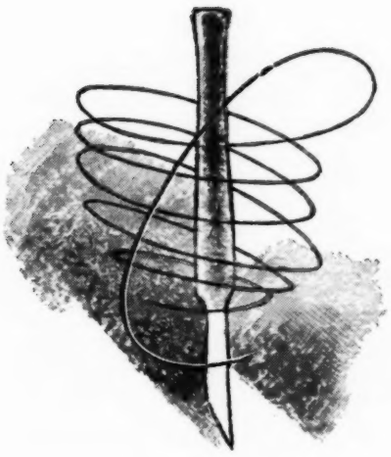

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susceptibility to shock during anesthesia and operation

tendency to liver damage

delay in wound healing

susceptibility to infection

Ravdin[1] also suggests that in the absence of frank signs of deficiency it is nevertheless reasonable to assume that deficiency exists whenever illness has been prolonged, particularly in the face of faulty dietary intake.

1. Ravdin, I. S.: Symposium on Nutrition in Surgery, Editorial, Am. J. Clin. Nutrition 3: 447-448 (Nov.-Dec.) 1955.

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NOTES AND QUOTES

RESEARCH grants approved by the Tobacco Industry Research Committee in the last year passed the $838,000 mark with the recent announcement of new grants to nineteen scientists and renewal of nine previous grants.

The new grants and renewals total more than $355,000, said Timothy V. Hartnett, Chairman of the Committee which sponsors research into tobacco use and health by independent scientists at recognized hospitals, laboratories and medical schools throughout the country. The Committee has set up a $1,000,000 research fund and has pledged more support as the need develops.

Grants are made by the Committee upon recommendation of a Scientific Advisory Board of nine noted doctors, scientists and educators who direct the Committee's research program and policy. Chairman of the Board and also Scientific Director of the Committee is Dr. Clarence Cook Little, Head of the Jackson Memorial Laboratory at Bar Harbor, Maine.

The Tobacco Industry Research Committee, formed in 1954 to support independent scientific research into tobacco use and health, is comprised of representatives of tobacco manufacturers and associations of growers and warehousemen. The Committee also provided a fund of $25,800 to sponsor fellowships in 1955 for medical students into basic and experimental sciences.

Two Yale faculty members, one a medical authority on bones and the second an engineering expert, have joined forces in an unique study of "bone engineering."

Under the direction of Dr. Charles O. Bechtol, Associate Professor of Orthopedic Surgery at the Yale School of Medicine, the research work is aimed at defining the characteristics of bone. The eventual goal is the perfecting of the bone splint as a means of healing severe fractures.

From the Yale School of Engineering, the project has drawn Henry A. Lepper, Jr., Associate Professor of Civil Engineering, to handle the technologic side of the experiments.

"Before we can study the problem of splints," says Dr. Bechtol, "we have to understand thoroughly the materials we are working with—bone and metal."

The bone splints in use today are made either of vitallium® or stainless steel, and are fastened to the bone by means of screws inserted in holes which have been drilled into the bone on opposite sides of the fracture. However, few doctors are convinced of the complete success of this type of operation, according to the Yale doctor. "For one thing, we've found in the engineering lab that drilling holes in bone can reduce its strength by as much as one third."

Supported by a grant from the Erdle-Prange Foundation, Dr. Bechtol and his associates are studying the how's and why's of bone and metal behavior. In the laboratory of the Civil Engineering Department, bones are subjected to a variety of pressures and tests, then drilled and subjected to the same tests again, to determine the strength and the weaknesses inherent in them. Yale civil engineers and medical researchers have also found that while any bone is more than strong enough for its job, it is extremely difficult to design and install a bone splint which can absorb the same stresses as the bone to which it is attached.

THE UNIVERSITY OF WISCONSIN'S pioneering role in many phases of medical research has been singled out for special attention in a survey of the last half-century of American medical progress conducted by The American Foundation. The foundation singled out the Wisconsin program of research as one best illustrating the contributions to medical progress that can be made by state universities. That all medical problems are problems of life chemistry was long ago pointed out by physiologists. Solution of these problems is therefore dependent upon basic research.

"Since medicine is rooted in biology and biology is becoming a molecular science," the report adds, "the scope of the medical research field is vast, including the nature and behavior of protoplasm, its molecular complexes, every aspect of growth and development from birth to death, every reaction to environment.

"Long before biochemical work assumed its present importance in biological and medical research, biochemistry in Wisconsin's agricultural college was aiding long-term programs in the departments of bacteriology and genetics.

"The impress of this department's major 40-year program, its original preoccupation with nutrition, its contributions to mineral

CONTINUED ON PAGE 58

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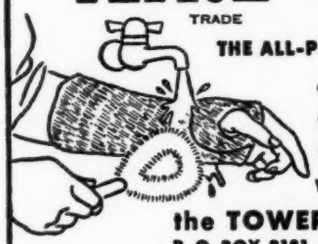

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metabolism, its studies of the role of trace elements in nutrition, its exploration of respiration may be found in many departments of the agricultural and medical schools."

Although nearly all of Wisconsin's major research projects that influence medical progress are described, some of those given particular attention are the fundamental work of bacteriologists on nutrition and metabolism of bacteria; work in plant pathology; genetics; human and animal nutrition; the work that led to the discovery of the vitamin B that cures pellagra; heart and cancer research; hormone physiology; the development of dicumarol; and work at the Enzyme Institute.

The American Foundation was established in 1924 by Edward Bok, an editor and writer, and since that time has undertaken broad studies of public questions, including those relating to high standards in medical education and medical care for the whole population.

A revolutionary plastic bag shows experimental promise of extending the period of storage of whole blood from the present twenty-one days to thirty-five, according to a Harvard blood expert.

Dr. John G. Gibson II told the American Association of Blood Banks that the bag eliminates the "turbulence and foaming" that occur when donor blood is drawn into a glass bottle, as is now the case.

"This turbulence is damaging to the red cells and contributes significantly to the reduction of their time of survival," Dr. Gibson declared.

Equipment of the type described has been developed and perfected by Fenwal Laboratories, Inc., Framingham, Mass.

At present it is a government regulation that whole blood can be kept for only twenty-one days. By extending the survival time to thirty-five days, the quantity of whole blood available in the country at any given time could be substantially increased, Dr. Gibson pointed out.

In addition, the patient receiving a transfusion would be getting a "healthier" blood.

FACED with ever-growing numbers of aging long-term patients, thirteen Veterans Administration hospitals in the East have taken the lead in activating an entirely new concept in federal medical treatment.

Known as the Intermediate Program, this plan will have the primary aim of providing active treatment rather than custodial care for long-term patients whose discharge is not yet advisable despite extended acute treatment.

The program stems from the increasing age level and attendant chronic illness of America's 22,000,000 veterans. Today, the average age of these veterans is thirty-eight with only 600,000 having reached the age of sixty-five. By 1960, it is estimated the average age will be forty-one with more than 1,780,000 over sixty-five.

To combat this problem, V. A. has extended the Intermediate Program for all-out care to at least thirty hospitals in the United States.

Absenteeism among employed persons is a subject of considerable concern to industry, according to a recent publication of the Research Council for Economic Security, Chicago. Illness absences alone, by the most conservative estimates, cause the loss of the services and production of one million workers every year, valued at approximately five billion dollars. If all data were available, the loss from absenteeism might be found to be as much as ten billion dollars.

"This is a problem," stated Gerhard Hirschfeld, Director of the Research Council, "which is in need of much more attention than it has received before. In addition to the production loss, there is the direct loss of wages and income for the workers and the cost of medical care. These are considerable even with the extensive protection of group sickness compensation, hospitalization, surgical insurance, and prepaid plans prevalent in industry today."

IN recognition of his long-time work in behalf of education of the public in fields related to medicine, John G. Searle, Chicago medical research and pharmaceutical manufacturing executive, was honored recently with fellowship in the Institute of Medicine of Chicago. This is an organization of leading physicians and has eighteen special medical and related societies as affiliates.

Conferring of the fellowship on Mr. Searle, President of G. D. Searle & Co., was made by Dr. Arthur R. Colwell at the Institute's 40th annual dinner.

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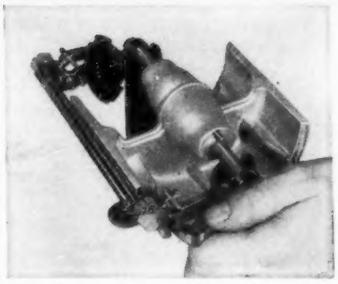

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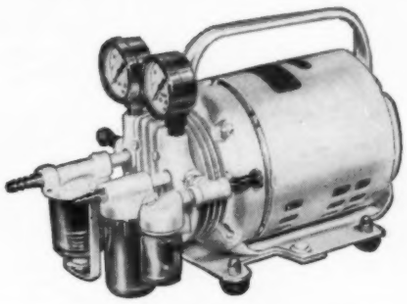

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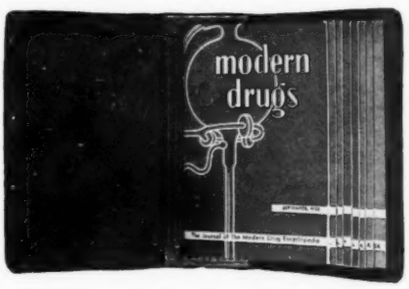

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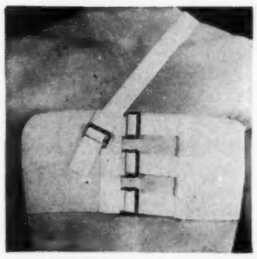

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NOTES AND QUOTES

Drs. Elizabeth L. Hazen and Rachel Brown of the Division of Laboratories and Research of the New York State Department of Health, Albany, have been awarded the first $5,000 Squibb Award in Chemotherapy in recognition of their discovery of nystatin, the first antifungal antibiotic safe enough for human use. The new drug was obtained by Drs. Hazen and Brown from a microorganism found in soil from a dairy farm in Fauquier County, Virginia. The antibiotic was first made available to doctors last year by E. R. Squibb & Sons under the name mycostatin,® and has been hailed by the medical profession as "filling an important gap in the range of drugs for chemotherapy." The drug is also being supplied to doctors in combination with tetracycline, a widely used broad-spectrum antibiotic, under the name mysteclin.

A new type of artificial respirator which "breathes" inside the patient was demonstrated to the Milwaukee Society of Anesthesiologists recently.

Dr. E. Trier Morch of Chicago invented the machine. He said it is one of the first machines which allows the operator to vary the pressure of air intake. Ordinary respirators apply pressure outside the patient.

This allows normal breathing artificially, Dr. Morch said.

One of the largest philanthropic contributions of equipment toward relief of human suffering from cancer became known recently when the Donner Foundation announced that it would donate twelve powerful Van de Graaff x-ray generators to as many hospitals and clinics for treatment of deep-seated cancer.

The twelve machines, valued at $68,500 each, are two million volt x-ray generators developed and manufactured by High Voltage Engineering Corporation, Cambridge, Massachusetts. Each institution receiving the equipment will provide necessary physical facilities and operators, and will pay for installation. Recipient hospitals will provide cancer treatment with the machine on a non-profit basis.

"Our primary goal in this program," said Robert A. Maes, Executive Vice President of Donner, "is to make advanced therapy available to the greatest number of cancer victims regardless of their economic status; hence our stipulation that treatment be provided at cost."

The Donner Foundation, Inc., was established in 1932 by the late William H. Donner, in memory of his eldest son who had died of cancer three years previously at the age of thirty-five. Until 1949 the Foundation's philanthropic activities were devoted exclusively to the fight against cancer. Most notable of those projects supported by the Foundation was the establishment and operation of more than twenty-five Cancer Detection Clinics throughout eastern Pennsylvania.

The Myasthenia Gravis Foundation, Inc., is currently making a wide distribution of a reprint of the Proceedings of the First International Conference on Myasthenia Gravis which it held in December, 1954, at the University of Pennsylvania. Copies are being sent to all Medical Libraries in the United States and in answer to requests from physicians in and out of the country.

An industry-wide public relations program for the entire drug industry, which will highpoint dramatic achievements in the health field, has been approved and underwritten up to $100,000 annually by the American Drug Manufacturers Association.

George F. Smith, President of Johnson & Johnson and Chairman of A. D. M. A.'s public relations committee, announced the association's Executive Committee decision and stressed that other drug associations and other organizations in the health field will be invited to participate in the program.

"We have kept the other organizations advised of our plans," said Mr. Smith. "However, in order to move ahead immediately, and pending the invitation to other drug and health groups to participate in both the policy and financial aspects, the A. D. M. A. has underwritten the industry-wide public relations program.

"It will be our objective to present to the people basic information about the research, developments and service wrought by those on the health team. The great achievements of the industry are unknown to the public generally and one of the objectives will be to inform the people of the nation of these accomplishments."

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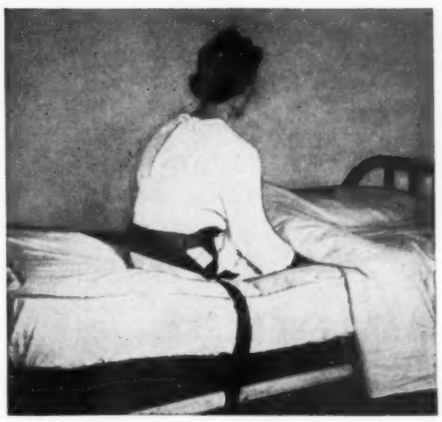

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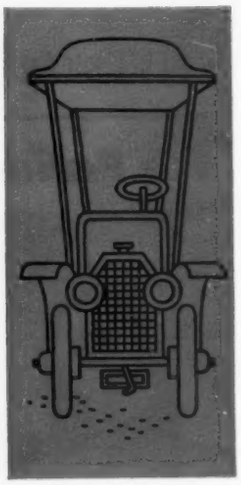

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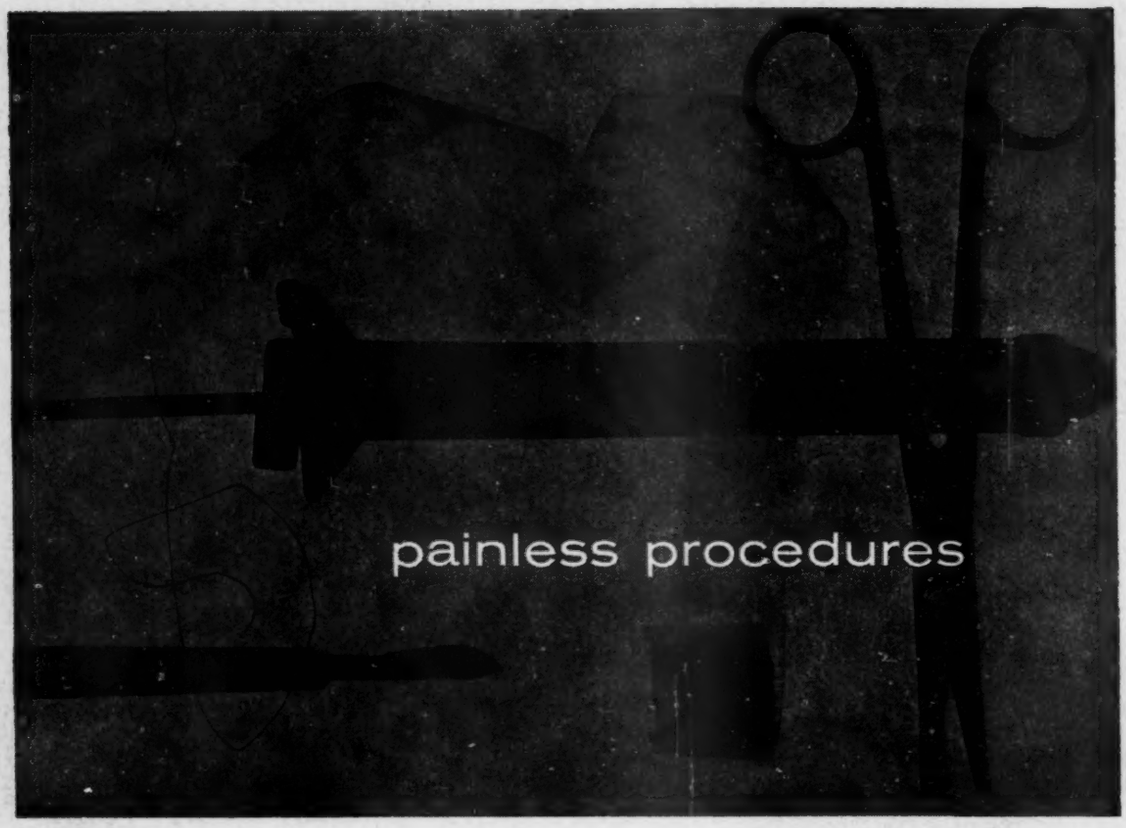

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